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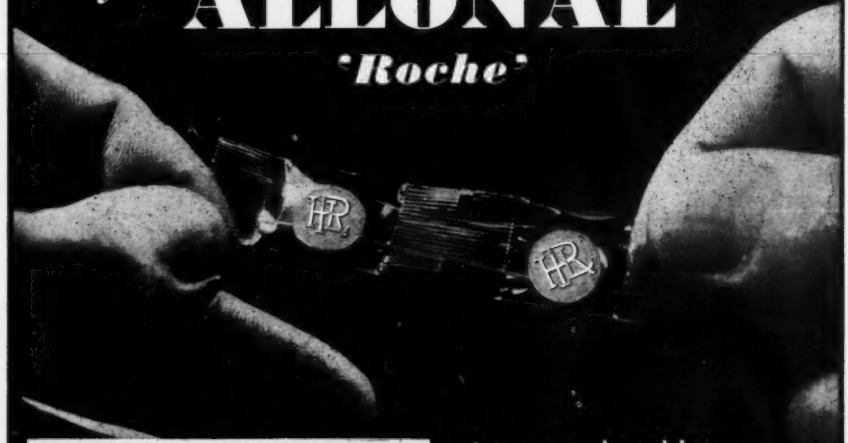

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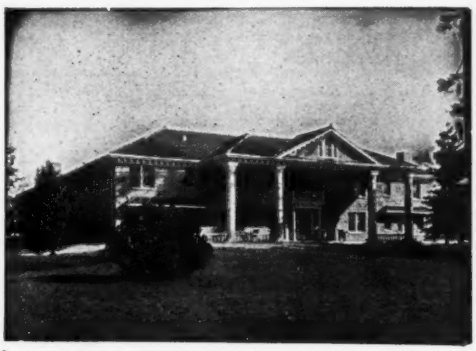

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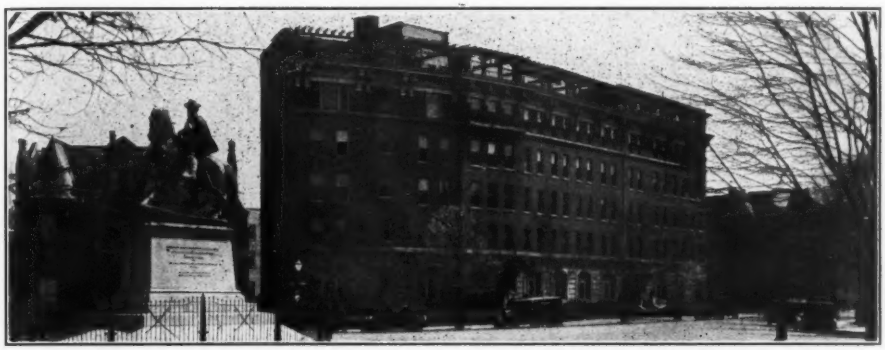

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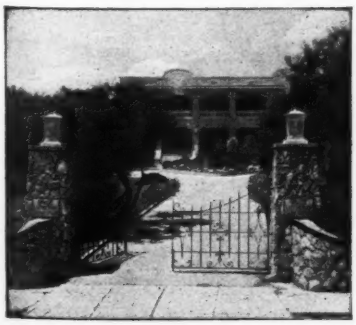

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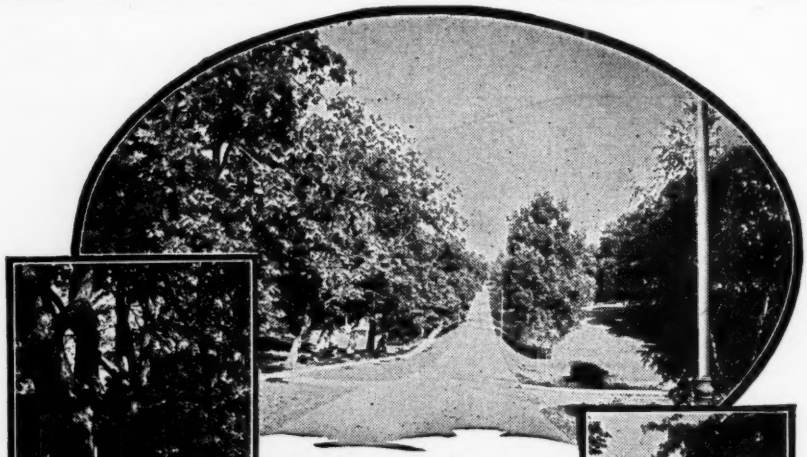

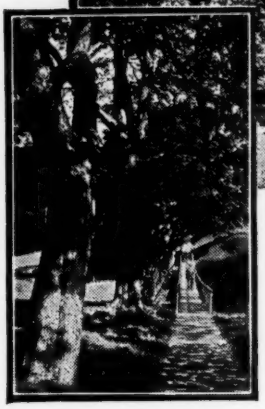

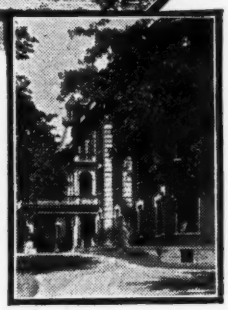

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CLINICAL-PATHOLOGICAL CONFERENCE*

I. A CASE OF SEVERE ANEMIA WITH CARDIAC MANIFESTATIONS

II. A CASE OF OBSCURE INFECTION

By LOUIS HAMMAN, M.D.,
Baltimore, Md.

The clinical-pathological conference has become deservedly popular in the curriculum of medical schools. There the student learns not only the methods of diagnosis, but also the value and the shortcomings of the methods. For after the diagnosis is discussed the clinical predictions are immediately tested by the incontrovertible evidence of the postmortem. No display of erudition, skill in argument or weight of prestige can prevail against the simple facts openly exposed. It is a hard discipline in clinical diagnosis and those trained in the school humbly acknowledge that "diagnosis is difficult and judgment often fallacious." The exercises are conducted in this manner. A member of the clinical staff is given the records of a patient's illness. These he studies and analyzes and from the facts there preserved draws the conclusions that seem warranted and predicts the lesions that will be found at autopsy. He may have seen the patient during life or he may not. The only restriction imposed is that he shall not have seen the autopsy nor have any knowledge of its revelations.

It will be impossible here to reproduce the deep interest and lively suspense these exercises arouse. The trenchant and penetrating remarks of the pathologist will be especially missed. However, I think the diagnostic discussion of any interesting case followed by autopsy control cannot be entirely without interest. The two cases I shall now consider were presented at recent conferences. I know what the autopsy revealed, but I have a written record of the clinical discussion made before knowing the results of the postmortem. This written record is the source of the diagnostic argument I shall give and I know you will trust me to play fairly.

*Read in Section on Medicine, Southern Medical Association, Twenty-Sixth Annual Meeting, Birmingham, Alabama, November 15-18, 1932.

I. A CASE OF SEVERE ANEMIA WITH CARDIAC MANIFESTATIONS

Case 1. The Clinical Records, Unit History No. 34438, Johns Hopkins Hospital.—A colored child, five years of age, was admitted to the Harriet Lane Home on March 19, 1921, complaining of cramp-like abdominal pain, marked constipation and occasional vomiting. She was fairly well-developed, but poorly nourished, and showed evidence of loss of weight. The cervical and inguinal glands were a little enlarged. The general physical examination otherwise was negative. The leucocytes were 34,000. The intradermal tuberculin test was negative. The urine contained a trace of albumin and a few casts. The roentgenogram showed an enlarged heart. The lungs were negative. The temperature varied from 99 to 100°, going once to 102°. It was thought that the patient's symptoms were due to digestive disturbances following improper feeding. Seven years later, on January 11, 1928, the patient went to the Orthopedic Department at the University of Maryland Hospital, complaining of pain in various joints and difficulty in walking. The impression gained from the examination was that the symptoms were due to arthritis. On March 6, 1930, she entered the University of Maryland Hospital on the medical service. The heart was found to be enlarged; a loud systolic murmur was heard at the apex, and there was, too, an indefinite diastolic murmur at the base. The patient had a profound anemia, and the blood displayed all the peculiarities that are characteristic of a sickle cell anemia. A diagnosis of postrheumatic valvular heart disease was made. One observer thought there was mitral disease, another that there was aortic stenosis, yet another that there was aortic insufficiency, and still another an adherent pericardium. The tonsils and adenoids were removed on March 20, 1930. On December 6, 1930, the patient entered the Johns Hopkins Hospital complaining of frequent attacks of rheumatic pain with fever, some pain about the heart, shortness of breath and frequent digestive disturbances.

The temperature was 98°, pulse 60, respirations were 22, and blood pressure was 122/56.

The patient was an underdeveloped child, looking younger than her age; fairly well nourished. There was marked pallor. A few of the superficial lymph

nodes were palpable, but there was no marked glandular enlargement. The eyes showed nothing abnormal. The nose and throat were negative. The lungs were clear. The heart was definitely enlarged; the apex beat was in the fifth interspace 9 cm. to the left of the midline. The apex beat shifted about 1 cm. with change of posture. The lung moved freely over the heart. The first sound at the apex was loud and tympanitic and was accompanied by a loud systolic murmur. A loud systolic murmur was heard all over the body of the heart. The second pulmonic sound was a little accentuated. The pulse was regular and equal at the two wrists. There was an occasional extrasystole. The abdomen showed nothing remarkable. The liver edge was felt at the costal margin. The spleen was not enlarged. There was bowing of the tibiae due to rachitic changes.

Course of Symptoms During Stay in the Hospital.—The blood showed a marked anemia and all of the peculiarities characteristic of sickle cell anemia. There was moderate leucocytosis. The urine had a low specific gravity, contained albumin, a few casts and many pus cells. The Wassermann reaction on the blood serum was negative. The x-ray showed evidence of old rickets in the bones and an enlarged heart. Great interest centered about the condition of the heart; the reports of the examination of the heart vary somewhat and the interpretations vary widely. A loud systolic murmur was heard by all. Some observers describe a peculiar sound heard at the base in diastole, differing, they say, from an ordinary diastolic murmur. The 'phthalein output varied from 40 to 60 per cent. The urea clearance was 24 and 26 per cent of the normal standard. On one occasion the patient was anuric for eighteen hours, and during this period had headache, nausea, epigastric pain and slight edema of the retina. At this time the nonprotein nitrogen rose to 53 mg. per cent. Following appropriate treatment these symptoms quickly disappeared. During the period she was in the hospital the patient had frequent attacks of epigastric pain with nausea and headache. From time to time there was a little tenderness and redness of the joints, often associated with a slight rise in temperature. The anemia did not improve during her stay in the hospital. She was discharged no better than when she entered.

After a brief period at home, the patient was readmitted to the hospital on April 5, 1931. She had been reasonably well until twenty-four hours before admission, when she began to have pain in the right thigh, which became progressively worse.

At examination her temperature was 100.4°, pulse 60, respirations were 24, and blood pressure was 115/55.

The patient's general condition was the same as on the previous admission. She complained bitterly of pain in the right thigh. There was no gross abnormality discovered to explain the pain. The examination was in all other respects as on the first admission, except that the liver edge now was felt 4 cm. below the costal margin. The spleen could not be felt.

Course of Symptoms During Second Period in the Hospital.—The patient was quite ill on admission and she grew progressively worse. She continued to complain of severe pain in the right thigh. There was tenderness over the thigh, but otherwise no abnormality could be detected. The heart rate and the breathing became progressively more and more rapid. A loud friction rub was heard over both backs, and later a loud, leathery friction rub was heard over the whole chest. Over the left lower lobe there was an area where the breath sounds were suppressed. The patient grew more and more deeply intoxicated, there was profuse sweating and she died five days after admission to the hospital.

LABORATORY EXAMINATIONS

BLOOD

	Hbg. Per Cent	R.B.C.	W.B.C.
December 6, 1930	35	2,010,000	4,650
March 11, 1931	37	1,780,000	18,800
April 9, 1931	46	1,970,000	25,160

Reticulocytes varied from 0.5 to 7 per cent.

A differential count made in the Clinical Laboratory showed:

	Per Cent
Juvenile neutrophils	7
Segmented neutrophils	56
Eosinophils	8
Basophils	0.5
Mature lymphocytes	25
Young lymphocytes	1
Monocytes	2.5

The red cells varied somewhat in size. A number of microcytes were present. Sickle-shaped and cigar-shaped red cells could be seen in the stained preparation. The majority of the red corpuscles were quite pale, although others seemed to be filled with hemoglobin. Two nucleated red cells were observed in examining 300 white cells. Platelets were numerous and a number of very large platelets were noted. Bleeding time was 2 minutes and coagulation time 5 minutes. Clot retraction time was one hour.

The Wassermann reaction on the blood serum was negative.

Specific gravity of the urine was 1.008 to 1.018; albumin either none or a trace. Occasionally a few casts were seen. There were no red blood cells, but many leucocytes.

Blood chemical findings were:

	N.P.N. Per Cent
December 9, 1930	40 mg.
January 6, 1931	53 mg.
January 9, 1931	36 mg.

Uric acid varied from 7 to 9 mg. per cent.

The urea clearance test was:

January 14, 1931 ..... No. 1—26 per cent normal standard
No. 2—24 per cent normal standard

Phenolsulphonephthalein test:

December 8, 1930	40 per cent
January 14, 1931	65 per cent

X-Ray Examinations.—December 10, 1930, feet and hands were negative.

December 18, 1930, the head shows irregular areas of bone erosion which may be due to one of the congenital anemias.

December 31, 1930, teleroentgenogram, MR 4; ML 8.8; T 22.5.

Electrocardiogram shows normal sinus rhythm. T-I and II very high amplitude, T-III inverted.

A biopsy was done, with excision of bone marrow from the left tibia. There was moderate hyperplasia of the bone marrow with sickling of mature red cells.

After dental examination, one infected tooth was extracted.

The temperature on the first admission varied from 98 to 100°, with occasional rises to 102°. On the last admission it varied from 99 to 102°.

DIAGNOSTIC DISCUSSION

Sickle cell anemia, which obviously is the disease from which this patient suffered, presents many interesting clinical manifestations, some of them illustrated in the record of this illness. The tendency of the red cells to sickle is a condition commonly found in the negro. Indeed, we have been accustomed to say it is found only in the negro, but recently instances of the anomaly have been reported in white families in which there was no reasonable ground for suspecting a negro progenitor.[1] It is estimated that from 6 to 8 per cent of the negroes in this country have this blood peculiarity. The condition is inherited and is transmitted as a dominant trait according to the law of Mendel. Fortunately most of those who have it remain well. At least they have no symptoms whatsoever that are attributable to it. Only a small number of those affected develop anemia and the other symptoms that frequently go with the anemia. What circumstances decide the transformation of latent sickling into active disease we do not know. The transformation is accompanied by a great destruction of red cells to which the anemia, and probably the other symptoms as well, are due. The conspicuous symptoms of the disease may be divided into five groups:

(1) The anemia which accounts for the weakness and prostration and at least in part for the breathlessness and palpitation.

(2) Abdominal symptoms, consisting of severe abdominal pain often accompanied by nausea, vomiting, rigidity of the abdominal muscles and obstinate constipation.

(3) Arthritic symptoms, consisting of pain in the extremities and tenderness and swelling of the joints, often severe and incapacitating.

(4) Cardiac manifestations; the heart is often enlarged and loud murmurs are heard.

(5) Chronic leg ulcers somewhat resembling syphilitic ulcers, which frequently come on late in the disease.

The patient now under consideration did not have leg ulcers, but she illustrates very well the first four groups of cardinal symptoms.

About the anemia we need say nothing. The blood findings are so characteristic there can be no error in the diagnosis of sickle cell anemia. The condition may at times be confused, on superficial examination, with hemolytic jaundice.

Abdominal symptoms were well marked in this patient. I doubt that the brief summary of the course of the illness sufficiently emphasizes this point. At times she complained bitterly of abdominal pain and often she was obstinately constipated. When we know a patient has sickle cell anemia these symptoms excite no particular comment because they so frequently go with the anemia. But if a patient comes under observation for the first time and is not conspicuously anemic our attention may be focused upon the abdominal symptoms and we may forget about sickle cell anemia. For the abdominal pain may be severe, the patient obstinately constipated, the abdomen tender and the walls as rigid as a board, and there may be fever and leucocytosis. Many patients are needlessly operated upon after a ready diagnosis of appendicitis. This past summer a young negro boy was brought to the hospital complaining of severe abdominal pain and constipation. The abdomen was tender and rigid, he had fever and the leucocytes were moderately increased. He was promptly operated upon, but at operation no abnormality was found. The following day it was discovered that he had sickle cell anemia of a moderate degree.

The joint symptoms are no less important than the abdominal symptoms. They give an appearance to the clinical picture of rheumatic fever and lead frequently to a diagnosis of rheumatism or arthritis. Joint manifestations were conspicuous in the patient under discussion. Before it was recognized that the child had sickle cell anemia a diagnosis of arthritis was made and even after the anemia had been determined the history of marked pain in the joints, coupled with the evidence of some lesion in the heart, led to the belief that the child had had rheumatic fever with endocarditis and pericarditis. I must confess that I find great difficulty in deciding what may have been the cause of the severe pain in the right thigh which came on suddenly six days before death and of which the patient complained so bitterly. There seems to be some connection between the pain and the sudden aggravation of symptoms that led so speedily to death. The pain was too disproportionately severe compared to the pain usually complained of by patients with sickle cell anemia for which no gross pathological explanation is found. I thought at first that the patient must have acute osteomyelitis of the right femur, but

1. Rosenfeld, S.; and Pincus, J. B.: Amer. Jour. Med. Sci., 184:674, 1932.

there was no swelling nor other local evidence of inflammation and the constitutional symptoms, especially the fever, were not so marked as they usually are with acute osteomyelitis. I feel convinced that the autopsy will disclose some local abnormality to explain the pain, although I am unable to suggest what abnormality unless it be acute osteomyelitis.

We come at last to consider the heart which is the point of special interest. The heart was somewhat, though not greatly, enlarged, a loud systolic murmur was always heard, a diastolic murmur was noted by a few observers, the heart shifted its position with change of posture but not so widely as some thought it normally should. Various diagnoses were made about the heart, which I shall tabulate, and I should like to impress upon you that these diagnoses were made not alone by house officers, but also by accomplished visiting physicians to the University of Maryland Hospital and to the Johns Hopkins Hospital. The cardiac diagnoses of seven observers read as follows:

(1) Mitral insufficiency and aortic insufficiency.

(2) Aortic stenosis and adherent pericardium.

(3) Mitral stenosis and insufficiency. Possibly adherent pericardium.

(4) Mitral disease and aortic stenosis.

(5) Probably mitral disease, but there may be no valve lesion.

(6) No valve lesion, but rheumatic myocardial disease.

(7) Refuse to make a diagnosis of valve disease in the presence of so severe an anemia. I suspect adhesive mediastino-pericarditis.

When there is such a wide difference of opinion among the experts what is a quite ordinary physician to say? It were better for him to hold his peace until the autopsy findings are revealed so that he might then enjoy poking friendly jibes at his confreres. But if he must speak out and give testimony to the faith that is in him then he may draw comfort from the thought that no matter what he may say, if he be wrong he will have erred in distinguished company and he may draw inspiration from reflecting upon two ironical aphorisms. First, when there is wide difference of opinion among competent observers about the exact nature of a valvular heart lesion, then it is altogether probable that the valves are not diseased. Second, when there is grave doubt whether the heart is diseased or not diseased, then in most instances it is not diseased. These aphorisms are especially applicable when a patient has severe anemia. This point cannot be too much stressed in diagnosis. In the presence of severe anemia murmurs are nearly always heard over the heart and frequently they are loud, harsh murmurs. The auscultatory signs may simulate mitral insufficiency or mitral stenosis, less frequently pulmonary or aortic stenosis. I am impressed by the fact that under these circumstances mitral stenosis is often erroneously diagnosed. I speak with feeling, having only recently fallen ignominiously into this error. The patient had pernicious anemia. The physical signs were closely imitative of mitral stenosis. She developed auricular fibrillation and died after a number of embolic phenomena had occurred. In discussing the case I pointed out that on the evidence of common experience a diagnosis of mitral stenosis was unjustified, yet I was so impressed by the signs and the course of the symptoms in this particular instance that willfully throwing discretion aside I committed myself to the diagnosis. Needless to add that the law of probability was sustained by the post-mortem. In sickle cell anemia signs, particularly auscultatory signs, of valvular heart disease occur frequently. This is true especially in children. Besides, the heart often is enlarged. Why it enlarges is difficult to explain because the valves are not affected and the heart muscle is not extensively diseased. Again and again a diagnosis of heart disease is made and again and again it is disproved at autopsy. In the patient we are considering the history of many previous attacks of pain in the joints with tenderness and swelling was pointed to by a number of examiners as evidence of rheumatic fever. This inference is unwarranted, since, as I have pointed out, such arthritic attacks are characteristic symptoms of sickle cell anemia. From these considerations ordinary prudence will dictate the prediction that in this instance as in so many others the autopsy will reveal a heart somewhat enlarged but otherwise normal.

There remains to be considered only the terminal pulmonary manifestations. As you may remember, on the last two days of life a loud friction rub was heard over both lungs and an area of suppressed breath sounds over the left lower lobe. These signs were interpreted to indicate the onset of a terminal bronchopneumonia, an event which in chronic disease is frequently the precursor of death.

Autopsy No. 11942.—The autopsy was performed thirteen hours after death by Dr. D. B. Remsen. The heart is described as perhaps a little large in comparison to the other organs. It weighs 280 grams. The epicardium and endocardium are smooth and glistening. The valves are all delicate and appear quite normal. The lungs are air-containing and show no gross lesions. The pleurae are smooth.

The spleen, however, is striking in appearance. It is small, round, with a deeply depressed scar at one point. It weighs only 45 grams and measures 5.5 x 5 x 3 cm. On section it is rather dark red in color and there are no visible Malpighian bodies.

The liver is swollen and pulpy and dark red in color. On section the lobules are blurred and gray patches are seen which apparently are about the portal veins.

The kidneys present a rather peculiar appearance. The capsules strip with some difficulty, leaving a finely granular surface. The little projecting nodules of kidney tissue are distinctly brown and the same red-brown color is seen on the cut surface. The glomeruli stand out as rather dark red dots against this yellowish-brown background. The cortical striations are indistinct. The larger vessels, both veins and arteries, are patent at the hilum.

The femur bone-marrow is dark red in color.

The brain appears to be normal.

Dr. A. R. Rich, who examined the microscopical sections, left a note that the spleen is entirely characteristic of the spleen in sickle cell anemia. "The pulp is everywhere filled with blood and this obscures, as usual in such cases, the abnormalities of the sinuses which occur around the Malpighian bodies. Numerous Malpighian bodies show the peculiar abnormal vascularity common in this condition, that is, they contain a multitude of capillaries throughout the lymphoid tissue. There are numerous rather extensive scars in the spleen, some containing calcium. These result from stagnation of blood in the overfilled pulp, and the organization of this stagnant blood. This sequence of events can be followed with perfect ease in other cases, but in the present case, at least in this one section, there is nothing to be seen but the scar. There are deposits of hemosiderin in the liver, spleen and kidney. The bone-marrow is hyperplastic. There are some obliterated glomeruli and small scars beneath the capsule of the kidney, but nothing indicating a progressive nephritis."

FINAL COMMENTS

The autopsy verifies the diagnosis of sickle cell anemia, but the correctness of this diagnosis was clinically assured. It fails to show any evidence of organic heart disease, as was correctly predicted. I am surprised that no local abnormality was discovered in the right thigh to explain the severe pain. We are forced to conclude that the pain was an unusual exaggeration of a symptom commonly complained of in sickle cell anemia. The absence of evidence of pleurisy or pneumonia or of any other pulmonary disease is disconcerting. All one can say is that the friction rub heard during life could not have been a pleural friction. The sound must have been due to something else and its significance was misinterpreted.

* * *

II. A CASE OF OBSCURE INFECTION

Case 2.—Unit History No. 31676, Johns Hopkins Hospital.—A white widow, giving her age as 51 but looking older, was admitted to the hospital December 8, 1930, complaining of fever and vomiting. She previously had been admitted on the Gynecological Service on three occasions. In 1917 she was operated upon for a draining sinus which followed a hysterectomy and bilateral salpingectomy performed in 1914. In 1920 she was treated for cystitis. In June, 1930, she was operated upon for a ventral hernia which followed cholecystectomy five years before. During this third admission it was noted that she had hypertension, the blood pressure being systolic 204 mm. of mercury, diastolic 100 mm. The patient left the hospital July 14, 1930, in good condition and she remained well until twelve days before her final admission, when she was suddenly taken ill with faintness, chill, high fever and vomiting. The fever continued and she was unable to eat anything on account of frequent vomiting; she could not retain even water. Six days before admission the mouth became sore and the tongue dry and cracked. Constipation had been marked.

Examination showed a temperature of 103.8°, pulse 94, respirations 18, and blood pressure 148/80.

The patient was a well nourished woman who obviously had lost weight. She was very apathetic, but was easily roused. She was deeply intoxicated. Her eyes were sunken and lips crusted. There was no herpes, no enlargement of the superficial lymph nodes, the thyroid was not enlarged. The left pupil was a little irregular and larger than the right, the result of an old injury. The movements of the eyeballs were normal. The eyegrounds showed nothing remarkable. The tongue was very dry and heavily coated. The lungs were clear. The heart was not enlarged, the sounds clear. The pulse was regular and equal at the two wrists, not dicrotic. The abdomen was prominent, there was a thick panniculus. No tenderness, no masses. The liver and spleen were not enlarged. One spot described as a rose spot was seen on the right side of the abdomen. The pelvic and rectal examinations were negative. Neurological examination was negative. There was no rigidity of the neck and no Kernig sign. There was an eczematous eruption over both buttocks.

Course in the Hospital.—The high fever, present on admission, continued and the patient gradually but progressively became more and more deeply intoxicated. The physical examination changed but little. Toward the end of the illness cyanosis became marked, respirations grew deeper and more labored and finally the signs of a terminal bronchopneumonia developed. The patient died in coma of circulatory failure twelve days after admission to the hospital and twenty-four days after the sudden onset of her illness.

LABORATORY EXAMINATIONS

BLOOD COUNT

	Hbg. Per Cent	R.B.C.	W.B.C.	P.M.N. Per Cent
December 8	90	5,740,000	9,720	71
December 11	86	4,280,000	18,000	79
December 18	66	3,820,000	14,200	88

Wassermann reaction on the blood serum was negative; flocculation test doubtful.

Blood cultures made repeatedly were all negative.

Agglutination tests with typhoid, paratyphoid, melitensis and typhus bacillus were all negative.

Urine examination showed specific gravity of 1.010 to 1.025, sugar 0, albumin a trace, and a few pus and epithelial cells in the sediment.

Urine culture was negative.

The stool always was positive for occult blood.

Cultures from the stools showed only colon bacillus.

Cultures from tonsils and nares were negative.

The temperature ranged from 102 to 105°, the pulse from 90 to 130, respirations from 24 to 40, occasionally going to 50. Blood pressure which on admission was 148/80, later rose, and once was as high as 218/75.

X-Rays.—June 18, 1930. Tele.: MR 3.5, ML 9.6, T. 26.1.

Chest: lungs clear. Heart and aorta within normal limits.

December 10, 1930. Chest: lungs clear

December 12, 1930. Chest: lungs clear.

December 18, 1930. Chest: bronchopneumonia left lung.

DIAGNOSTIC DISCUSSION

This is the story of a patient taken suddenly ill with high fever and prostration, becoming progressively more and more deeply intoxicated, finally developing bronchopneumonia and dying twenty-four days after the onset of the illness. Although very thoroughly studied during the twelve days she was under observation in the hospital, no additional data were obtained to throw light upon the nature of the infection. The symptoms are not characteristic of any of the ordinary infections with which we commonly deal. It is impossible to fit them accurately into any of the usually accepted diagnostic categories. Under these circumstances we must consider all the possibilities that come to mind and carefully weigh the probabilities for and against each. In this way we may perhaps suggest the correct diagnosis, although it is needless to say that from the given data it would be impossible to make the correct diagnosis with assurance. Let us, then, consider the various possibilities.

(1) *Typhoid Fever.*—When the patient entered the hospital the possibility of typhoid fever was at once suggested. But as the investigation proceeded no single bit of evidence was obtained to support the suggestion. One or two spots were observed which might perhaps have passed as rose spots, but these soon faded and no fresh ones appeared. The spleen was not enlarged. The leucocyte count was a little elevated and the polymorphonuclear cells a little increased. Both the fever and the pulse rate were high. Careful bacteriological studies failed to reveal organisms of the typhoid group in the blood, urine or feces. The last named feature is almost decisive, for with modern methods of study typhoid bacilli are almost always found in typhoid fever. The failure to find them after diligent search is the strongest possible evidence that the disease under consideration is not typhoid fever.

(2) *Pneumonia.*—The illness has none of the features of an ordinary lobar pneumonia. That need not even be considered. It is barely possible that the symptoms may have been due to an unusual form of bronchopneumonia, but that too is most unlikely. For there were no manifestations of bronchopneumonia until near the end of the illness. Up to that time the lungs had been clear to physical and to roentgen ray examination. The course of events clearly marked the pneumonia as a terminal bronchopneumonia coming at the end of some other severe infectious disease.

(3) *Septicemia.* — The numerous negative blood cultures exclude the possibility of the illness being due to a streptococcus, staphylococcus, pneumococcus or influenza bacillus sepsis. These organisms repeatedly sought for could not have escaped detection had they been the cause of so severe an infection. Among the bacteria less often causing sepsis the gonococcus and the meningococcus must be considered. In gonococcus sepsis the gonococci usually settle upon the heart valves producing the clinical picture of acute or malignant bacterial endocarditis. However, whether they settle upon the heart valves or not gonococcus sepsis is always associated with a high leucocytosis, which the patient under consideration did not have. Meningococcus sepsis in the absence of meningitis is uncommon, but now and again it does occur. As a rule patients with meningococcus sepsis are not so ill nor so deeply intoxicated as was the patient being reported upon, and in the instances of this condition which I have seen either the organism was eventually cultivated from the blood or the patient developed meningitis. In meningococcus sepsis also there is a marked leucocytosis. Perhaps one is not justified in excluding sepsis from further consideration, but certainly the evidence is strongly against it.

(4) *Tuberculosis.*—Miliary tuberculosis may produce a picture resembling this. As a rule, miliary tuberculosis is not so rapidly fatal, it does not produce such extreme symptoms of intoxication and manifestations of local tuberculous disease, for instance, meningitis, pericarditis, tuberculosis of the lungs, soon or late appear. The only local manifestation this patient exhibited was a terminal bronchopneumonia. The symptoms of miliary tuberculosis vary with the point of entry of the tubercle bacilli into the circulation. If the primary tuberculous focus

discharges into the thoracic duct or directly into a vein the tubercle bacilli are carried first to the lungs, where most are filtered out, only a small number passing on to the left side of the heart, from whence they go through the arterial circulation to liver, spleen, kidneys and other organs. Under these circumstances the clinical manifestations correspond to the anatomical distribution of the tubercles. If the primary tuberculous focus discharges into the pulmonary veins, then the tubercle bacilli go first to the organs where most of them lodge and only a relatively small number goes to the lungs. Under these circumstances the pulmonary symptoms are inconspicuous. If the patient being considered really had miliary tuberculosis, then it must have been of the last named type, because until the end of the third week of her illness there was no evidence of any pulmonary involvement. Miliary tuberculosis is the diagnosis that was favored while the patient was under observation in the hospital, but to show what little confidence was felt in the diagnosis it is followed by a question mark.

Of all the usual infections the clinical picture looks more like tuberculosis than any of the others, but even so the resemblance is not close enough to permit confident identification. Therefore, we must go further and call up unusual diseases which we see only occasionally, hoping the clinical features may match theirs more closely.

(5) *Typhus Fever.*—Sporadic cases of typhus fever come to our notice now and again. However, this patient had none of the symptoms that are characteristic of that condition. Sporadic typhus is usually a mild disease from which most patients recover. The eruption is a constant and important symptom. Most cases give an agglutination reaction with the typhus bacillus.

(6) *Malta Fever.*—The prevalence of infection with the *Bacillus melitensis* has been sufficiently emphasized and we now look for evidence of its presence in all patients with obscure fever. However, the symptoms of this infection are usually mild and the course prolonged and irregular. In this country acute fulminant cases of Malta fever are rarely seen. The diagnosis is difficult because of all infections it shows fewer localizing manifestations than any other. In the case we are now discussing the severity of the symptoms, the progressive uninterrupted course, the absence of *Bacillus melitensis* in blood culture and the negative agglutination tests are the points strongly against Malta fever.

(7) *Tularemia.*—I confess I would not think of suggesting tularemia had it not been for a recent experience.

A colored man (Unit History 40943) entered the hospital with high fever and symptoms of profound intoxication. Two weeks before he had apparently caught cold and had fever and prostration. Four days after this he had a violent chill and from then on grew progressively worse with high fever, prostration and cough and expectoration. The physical examination revealed no abnormality except the signs of bronchopneumonia in the lower lobe of the left lung. On the third day in the hospital a greatly enlarged right epitrochlear gland was discovered and careful inspection of the right hand then revealed an area of infection in the right thumb. Inquiry disclosed that shortly before the onset of his illness the patient had dressed rabbits and in breaking off the legs had wounded the right thumb. The diagnosis of tularemia seemed assured. The blood serum agglutinated the *Bacillus tularense* in a dilution of 1 to 160. The organism could not be grown from the blood nor from the pus obtained by incising the thumb nor from the fluid obtained by aspirating the enlarged epitrochlear gland. The patient died six days after entering the hospital.

If we exclude from consideration the evidence of the portal of entry of the infection and the enlarged regional lymph nodes then the two cases otherwise correspond exactly. Both were taken acutely ill with all the symptoms of a severe infection; the examination in both showed no abnormality except a terminal bronchopneumonia; in both the infection was overwhelming and the symptoms of intoxication progressed steadily and rapidly. One patient died twenty days after the onset of the illness, the other twenty-four days after. In the instance of the patient we are now considering, no agglutination test with the *Bacillus tularense* was made because at the time this possibility was not considered. Were we able to demonstrate a portal of entry of the infection with swelling of the lymph nodes draining the area, the evidence for tularemia would be complete. But it is well known that in many instances of tularemia a portal of entry cannot be demonstrated, these instances being spoken of as the typhoid form of the disease.

Summing up all of these considerations, I should say that the patient must have had either tuberculosis or tularemia, and in view of my recent experience I believe it was tularemia.

Autopsy No. 11764.—The autopsy was performed twenty-two hours after death by Dr. F. B. Kindell. The lesions which seemed to be of importance in the final illness were limited for the most part to the lungs

and lymph nodes. The heart was quite normal. The left pleural cavity contained about 150 c. c. of bloody fluid, and over the lower lobes of both lungs there was a fibrinopurulent exudate. Dr. Kindell notes that on section of the lung there were areas in the posterior part of the right lower lobe which suggested caseous pneumonia, few in number and rather small. Near the margin of the left lower lobe were found two nodules beneath a sunken area in the pleura, which on section were rather firm and pigmented. The peribronchial nodes were enlarged and contained caseous areas. The whole picture seems to have impressed him at the time as being tuberculous in origin. In addition to these changes he found rather large masses in the spleen and liver which he described as tubercles. Near the hilum of the liver there was a group of softened lymph nodes which formed a mass about 5 x 2.5 cm. On section, necrotic tissue was seen with some liquefaction, and in one or two places there was an extension forming little abscesses in the surrounding fat.

The spleen was enlarged and soft; it weighed 320 grams. The gallbladder contained eight stones with an average diameter of about 1 cm. The kidneys appeared to be normal. The aorta showed a considerable degree of arteriosclerosis, with elevated plaques about the intercostal arteries.

Dr. W. G. MacCallum's comment on the microscopical sections states that the lung, liver, lymph nodes and spleen show areas which at first sight look like an acute tuberculous necrotizing process, soft tubercles which usually show a great number of bacilli and fragmentation of nuclei. However, no actual tubercles are seen and there are no lesions such as intestinal ulceration or bronchial changes, which might be regarded as typically tuberculous. Most important of all, no tubercle bacilli are found on careful staining and after thorough search. Stains for other bacteria, including the Nicolle tannin methylene-blue stain for glanders, reveal no organisms.

Cultures showed no growth and a guinea pig which was inoculated with material from one of the nodes is still alive and well four months after inoculation. This may be due to the fact that the lymph node was kept for some time in the icebox before inoculation.

A later autopsy, No. 12355, which was definitely identified as tularemia, shows lesions identical with those described in this case, and there seems very little doubt that the present case is one of tularemia.

FINAL COMMENTS

This case illustrates how important it is always to think of unusual conditions when the clinical facts do not fit smoothly into commonly accepted categories. However, tularemia is no longer a rare disease and the possibility of obscure fever being due to tularemia should always be considered. The feature I wish especially to emphasize is how closely the anatomical lesions of tularemia simulate the lesions of tuberculosis. I have no doubt that many cases of tularemia are buried in pathological archives under the heading tuberculosis.

AN OPERATION FOR LATE REDUCTION OF THE SEMILUNAR BONE*

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The semilunar bone, when left dislocated for more than two or three weeks, is difficult to replace and maintain in a completely reduced position. Excision generally is practiced. It is undeniable that removal of this keystone of the radio-carpal joint is mutilating and disorganizing to such a complex packet of articulations as the carpal bones. The ideal treatment, therefore, would be that of reduction rather than excision. The bone is excised principally for two reasons: first, because it is easier to do so, and is less traumatizing than replacement in late cases; second, it is claimed that the results of excision are just as good as that of reduction.

In a former article† a summary of the opinions in the literature showed that the results of late reduction were often satisfactory, but often enough unsatisfactory to encourage recommendation of excision. In the former article it was pointed out that the 5 cases reported, and the cases reported by others, were characterized by a neurospastic fixation reaction which was mild in some cases and severe in others. It was observed that where the phenomena of neurospastic fixation occurred to a severe degree, the functional recovery was consistently unsatisfactory. It is reasonable to assume, then, that if a late reduction was attempted in a case where the symptoms were severe, failure to secure satisfactory function probably would be attributed to traumatic arthritis from excessive trauma in accomplishing reduction. In such a case excision would give no better result. If the case belonged to the type of mild symptoms of pain, muscle spasm and contraction, success for the procedure would be acclaimed whether it was that of excision or reduction. Taking into consideration, then, that ultimate recovery may be satisfactory or unsatisfactory, depending upon the type of mild or severe extent of neurospastic fixation deformity, both excision and replacement in late cases offer about the same prospects as far as the individual technic is concerned. Of the two, replacement, if it can be accomplished, would seem far superior to excision be-

*Read in Section on Bone and Joint Surgery, Southern Medical Association, Twenty-Sixth Annual Meeting, Birmingham, Alabama, November 15-18, 1932.

†McBride, Earl D.: Dislocation of the Semilunar Bone. Neurospastic Fixation of the Hand, a Deformity Characteristic of the Injury. Arch. Surg., Feb., 1927.

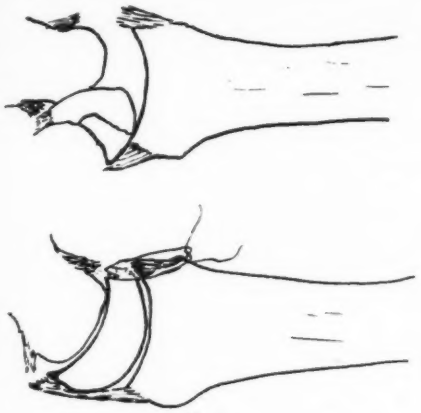

Fig. 1

Bridle operation for restoring posterior ligament fixation after reduction of dislocated semilunar bone.

cause it has the additional advantage of restoring normal anatomical relationship.

The chief cause of disappointment after late reduction is failure completely to reduce and retain the semilunar in normal position. At operation the bone may appear to be restored to proper relationship with the other articulating surfaces, but an x-ray examination afterwards may reveal a partial subluxation forward; a position that will limit flexion and perpetuate pain on motion. In such a position the head of the os magnum lies dorsal to the semilunar, and articulation with the scaphoid and cuneiform bones becomes insecure because of the narrowness of the dorsal one-third of the semilunar. Excision would be preferable to such an incompletely replaced position.

A procedure which assures retention has been used in four successive cases with better end-results than could have been secured by excision. It consists of firmly anchoring the dorsal horn of the semilunar to the radius, thus restoring the effect of the dorsal ligament. When secured in this manner the bone is in an attitude of complete dorsiflexion which blocks its tendency to slip partially forward.

Success of the operation for late reduction depends upon:

(1) Complete freedom of the ligamentous contraction and fibrous tissue attachments of the anterior horn to the radius.

(2) Complete removal of fibrous tissue in the cavity formerly occupied by the semilunar, and abnormal attachments of the os magnum.

(3) Security of the semilunar in its normal articular bed, so that it cannot slip or rotate forward.

The first two requirements may necessitate both a dorsal and anterior incision, especially if the case is of old standing, because complete release of the anterior ligament contraction and the removal of scar tissue is essential to complete reduction.

The third requirement is accomplished by securing the dorsal horn of the reduced semilunar to the dorsum of the radius in its completely dorsiflexed position. The interossei ligaments to the scaphoid, cuneiform and os magnum being torn, these bones must be brought to the position of dorsiflexion to meet the alignment of the fixed semilunar. If they are left in the flexed position, all three bones will be rotated dorsal to the dorsal horn of the semilunar.

Details of Operation.—An incision about 1½ inches long is made on the dorsum of the hand immediately to the ulnar side of the extensor carpi radialis. The vein and nerve, together with the extensor indicis tendon, are retracted to the ulnar side and the extensor carpi radialis to the radial side. The fibrous tissue in the bed formerly occupied by the semilunar is excavated and a smooth curved periosteal elevator passed forward to free the anterior horn contraction from the radius. The bone which lies in a plane entirely anterior to the carpal bones is then pried into position by a Davis skid or a similar instrument. If it cannot be reduced

Fig. 2

Characteristic neurospastic fixation deformity of the hand, due to semilunar dislocation. Either excision or reduction will fail to give satisfactory result where severe stiffness occurs.

Fig. 3
Semilunar dislocation, three months' standing; usually considered irreducible.

without too much trauma it is better to make an anterior incision immediately to the ulnar side of the palmaris longus, with its center over the radiocarpal articulation. The group of flexor profundus tendons are retracted to the ulnar side and the median nerve and palmaris longus tendon to the radial side. Care is used not to damage tendon sheaths or nerves.

Complete reduction may be recognized by the contour of the dorsal horn. When it is entirely reduced the exposed dorsal surface of the semilunar has an area about the size of a dime. If the bone has a tendency to pull forward or is not entirely in alignment with the transverse plane of the wrist, the anterior ligament is not entirely free. It is rather difficult to make a hole in the bone, so that with a curette a crevice is made in the anterior face of the dorsal horn of the semilunar, creating a hook into which a strand of No. 00 plain catgut is inserted and sutured to the dorsum of the radius. The semilunar thus is checked in full dorsiflexion to the radius as the bit and reins bridle check a horse.

The first case is one in which the operation was planned for removal of the bone, but instead reduction was accomplished. Relapse occurred because the bone was not anchored. In a second operation, through the dorsal route, the bone was firmly anchored in dorsiflexion by the newly devised method and the final results were very satisfactory. Later cases handled in a similar manner have given such satisfactory results that reduction rather than excision is thought to be the choice of procedures.

CASE REPORTS

Case 1.—H. W., age 21, was a white truck driver. While cranking his engine, his left hand was forced suddenly backward. The condition was treated as sprain for eight weeks. There was continuous pain and moderately severe stiffness. X-ray revealed the dorsal horn of the semilunar rotated completely anterior to the head of the os magnum.

An incision one and one-half inches long was made on the palmar aspect immediately to the ulnar side of the palmaris longus. Using care not to open the tendon sheaths, the flexor profundus tendons were retracted to the ulnar side and the median nerve and palmaris longus to the radial side. The semilunar was found lying in line with the longitudinal axis of the

Fig. 4
Semilunar dislocation of three months' standing; completely reduced. X-ray was made one year after reduction. No evidence of absorption.

Fig. 5
Old unreduced semilunar, excised. Shows marked loss of stability to carpal articulations.

radius, its concave surface facing the palm. The anterior horn was bound firmly to the radius but the anterior semilunar os magnum ligament was completely severed so that the head of the os magnum was entirely dorsal to the semilunar. The scaphoid-semilunar ligament and the semilunar cuneiform ligaments were severed except the portion attached to the dorsal horn. The attachments to the radius were freed sub-periosteally and the posterior horn of the semilunar was forced backward, thus reducing the bones to what seemed to be normal relationship. A plaster splint was used with the hand in slight palmer flexion.

X-ray the second day after operation revealed relapse. Two weeks later, a second operation was performed. A dorsal incision was used; the semilunar was levered into normal position; a notch was made in the anterior horn, deep enough to hold a strand of No. 00 plain catgut. The semilunar thus "bridled", the catgut was firmly secured to the fascia and periosteum on the dorsal of the radius. A Palmer plaster splint was applied with the hand in dorsiflexion. The reaction was moderately severe for three weeks. Eight months later he was doing ordinary manual labor.

Case 2.—H. G., age 19, a school boy, injured the right wrist seven weeks previous to admission. X-ray showed a rotation forward of the semilunar bone. He had 75 per cent dorsiflexion but no power of flexion. His fingers were slightly flexed with inability to extend. Pain was moderate. An incision two inches long over the dorsum of the right wrist immediately internal to the extensor indicis. The bone was freed from contraction of the naterior ligament and levered into proper position. A notch was made in the dorsal horn and the bone "bridled" to the radius with No. 00 plain catgut. Physiotherapy was advised but the patient did not return after the twelfth day. He was seen one year later, working at a dairy, milking several cows twice daily. He apparently had no disability whatever.

Case 3.—W. B., age 32, white, a tankbuilder, 28 days previous to reduction fell 30 feet into a tank. X-ray revealed the head of the os magnum to be displaced dorsal to the semilunar which was rotated on the transverse axis but remained in the longitudinal plane of the radius. The scaphoid was fractured, the proximal fragment being rotated toward the lateral border of the radius. A chip fracture of the triquetrum was present; also a fracture through the articular facet of the outer tip of the radius. The disarrangement of the carpal bones was interesting in that the semilunar, although rotated on its transverse axis as usual, remained in the proper longitudinal axis, while the carpal bones attached to the digits, that is, the cuneiform, os magnum, and trapezoid were shifted medially to the thumb side, leaving the semilunar, pisiform, and triquetrum in proper alignment with the ulna. There was considerable swelling of the wrist with inability to straighten the fingers. Pain was complained of, but it was not severe, and there was no evidence of marked circulatory or neurospastic changes.

A dorsal incision only was made. The semilunar was found rotated forward on the transverse axis. It was replaced by leverage and anchored in full dorsiflexion by catgut bridle to the radius. The triquetrum bone was completely excised because of a crushing fracture. A fragment of the radius was replaced. It was splinted in dorsiflexion ten days; then active motion and massage were employed. Two months later, he had 50 per cent dorsiflexion, 75 per cent palmar flexion, 75 per cent rotation and lateral motion. Six months later, he had moderate thickening of the wrist, full supination and pronation, with 75 per cent of normal grip, 10 per cent limitation of dorsiflexion and 5 per cent limitation of palmar flexion. On final examination ten months after operation, he had returned to his work of trucking. He had residual limitation of dorsiflexion of about 10 per cent and palmar flexion of 5 cent cent. Use of the fingers and hand was natural and practically unlimited except in strength which was about 25 per cent less than normal.

Case 4.—W. W. B., age 38, a white farmer, injured the right wrist cranking a car two months previous to operation. He had been treated for sprain. The wrist was swollen, the fingers partially flexed with inability to extend them. Dorsiflexion was limited 75 per cent, palmar flexion was limited 85 per cent. His grip was very weak, as he could barely close his fingers into the palm. X-ray showed the semilunar rotated and displaced forward.

A dorsal incision was made, and the semilunar loosened freely and levered into position by prying dorsalward on the anterior horn while traction was made on the hand. It was reduced with comparatively little trauma but had marked tendency to rotate forward. A dorsal horn notched and with bridle rein suture, the semilunar was fixed in full dorsiflexion to the radius. The hand was put in dorsiflexion, in plaster splints. The reaction was marked for four weeks. Seven months after operation, the appearance of the wrist was normal; he had full palmer flexion, dorsiflexion and lateral motion. His ring finger does not close easily apparently

because the operative scar had grown to the extensor tendon sheath. He milks five cows every morning and night and does his farming duties without favoring the wrist. He notices soreness sometimes after a hard day's work.

Case 5.—O. E. H., age 34, white, a plasterer, nine weeks previous to operation injured his right wrist while cranking a car. He was treated eight weeks for sprain. He cannot fully extend his fingers or close them. Pain is very moderate. There are severe neurospastic symptoms. X-ray shows a typical dislocation of semilunar bone.

A dorsal incision was made. The bone was firmly bound to a position anterior to the os magnum. It was reduced by leverage and bound to the radius in full dorsiflexion by a bridle rein suture through a notch in the dorsal horn of the semilunar. In this case the scaphoid-os magnum relationship was disturbed in that the scaphoid was found displaced 50 per cent dorsalward. The position of dorsiflexion of the hand restored alignment completely. The immediate postoperative reaction was quite severe. Six months after operation, was complaining of soreness and inability to return to plastering. One year after operation, information was obtained that the patient had returned to work. Examination was not made.

Case 6.—J. M. H., a truck driver, age 40, white, one week previous to admission, injured his right wrist while cranking car. His wrist was splinted. Considerable pain and swelling followed. On admission the wrist was found slightly flexed. He was unable to fully straighten his fingers and could not extend his wrist beyond a straight line. The semilunar was displaced forward.

A dorsal incision was made. The tendons were retracted in the usual manner. The wrist was forcibly flexed with an elevator under the semilunar. Replacement was rather easy because the injury was seen early. The semilunar had a tendency to slip forward of the transverse axis. It was fixed with a bridle loop to the radius. Reaction was moderately severe for ten days. Swelling gradually subsided. At last examination, seven months following injury, he is able to work at usual occupation. There is slight tenderness on forcible dorsiflexion.

DISCUSSION (*Abstract*)

Dr. O. P. Board, Birmingham, Ala.—Semilunar dislocations are often complicated by fractures. The question of removal or replacement of a semilunar depends upon the damage to the bones and blood supply at the time of injury. If at operation much injury is found to the semilunar or is caused by the replacement it is wiser to remove it. Otherwise experience has shown that traumatic osteo-arthritis develops, with pain and impairment of motion.

Dr. Philip D. Wilson, Boston, Mass.—I have never attempted to replace a semilunar bone that had been dislocated for a period of more than four weeks. Prior to this time I consider reduction infinitely preferable to excision. After four weeks, however, the cartilaginous degeneration has usually progressed to such a point that recovery of function is scarcely to be expected, and on the whole I have supposed that excision gave a better result than replacement.

Dr. McBride's experience seems to indicate that the semilunar bone, if replaced, is still capable of function at long periods after injury, and this would indicate that we must revise our ideas on this matter. When there is a combination of fracture of the scaphoid in association with dislocation of the semilunar, reduction should be urged when the patient is seen within a short time after injury. At a later period it is quite a problem what is the best treatment. Excision of both scaphoid and semilunar yields a useful wrist, but rarely one that is entirely normal. Reduction of the displaced bones would necessitate a bone grafting operation on the scaphoid, and while this has given good results in my experience in isolated fractures of the scaphoid, I have not attempted it when there has been an associated dislocation of the semilunar. It may be that the best treatment in such cases would be to perform an arthrodesis between the os magnum, scaphoid and semilunar as described by Thornton some years ago.

Dr. W. B. Carroll, Dallas, Texas.—We have never attempted late reduction of a semilunar as described by Dr. McBride because we have believed so much trauma would be necessary that final function would be greatly impaired. Even in early cases with open reduction there has been in our limited experience considerable difficulty in replacing the bone. However, once it is replaced there does not seem any marked tendency for it to be displaced, especially if the wrist be kept in flexion.

This work of Dr. McBride's with functional results as shown in his pictures lends encouragement to our attempting better restoration in wrists which we had been accustomed to consider permanently impaired in function.

Some experimental work has been done by Dr. Eggers, of Galveston, on the mechanism of semilunar dislocations. A combination of dorsiflexion and medial deviation of the hand is necessary, he has shown, for avulsion of the semilunar. This work is not yet published.

Dr. E. Laurence Scott, Birmingham, Ala.—I treated a fractured scaphoid a long time ago and I had reason to think I could put the two pieces of scaphoid together. I still have my patient. I saw him every three or four months for a long time and still see him occasionally.

Dr. McBride (closing).—Judging from the discussion of my paper the argument seems to stand in favor of replacement. This, however, is different from advice gained from opinions expressed in literature. Any disturbance of anatomical relationship of the carpal bones will create disturbance in function. I have satisfied myself that the semilunar can be replaced even as late as three or four months after injury. In most instances the disability has been very much less than the literature would lead one to believe, and I do not intend to remove any more semilunars unless it is absolutely necessary. In the early cases the bone is so loose that it is tempting to remove it, just as Dr. Key says, but I would very strongly oppose this procedure. I have not, as yet, found any case which developed Keinbock's disease or any evidence of atrophy following surgical replacement.

DENERVATION OF THE URETER: A CLINICAL STUDY*

By LAWRENCE R. WHARTON, M.D.,
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One of the most distressing situations that the physician encounters is persistent ureteral colic. Fortunately, such cases are not extremely common for it is usually a fairly simple matter to detect the cause of the discomfort and bring relief. There remain, however, exceptional cases which baffle our diagnostic efforts and resist all known methods of treatment. It is this particular group that we are now considering.

Taken as a whole, these cases have certain definite characteristics. The outstanding feature is that the pain is entirely out of proportion to the demonstrable pathological findings and persists after they have been corrected. There may be a slight hydro-ureter or hydronephrosis, hypermobility of the kidney, a history of pyelitis or we may suspect that the pain is secondary to some irritating lesion in the tubes and ovaries. If the diagnosis of ureteral stricture is made, the pain returns with increasing severity after each course of dilatations. It is again characteristic that there is no evidence of actual persisting ureteral constriction or obstruction, because a number eight or nine renal catheter passes to the kidney without any difficulty.

In the majority, there is no hydronephrosis, no hydro-ureter, pyelitis or significant impairment of renal function. Under these circumstances, therefore, one faces the proposition of increasing ureteral pain which has no satisfactory organic explanation and which one is unable to relieve. Occasionally, if the pain is unilateral, in desperation the kidney is removed; and this is always curative, as far as that side is concerned. Unfortunately, if anything happens later to the remaining kidney or ureter, the seriousness of the dilemma is increased. In this report we shall refer to three such cases.

It is in hopeless situations such as this that we have been using ureteral denervation since 1924, when Dr. Walter Hughson and I devised this operation. We have been doing this on the assumption that the pain is due to ureteral spasm and that it may be the persisting aftermath of an old urinary infection, a mechanical disturbance, or a pathological process in some contiguous organ. Since 1924, I have performed the operation upon 8 women (9 ureters). And because the results have been gratifying and have altered the whole outlook on life for some of these women, I am herewith recording my experiences. Fortunately, due to the courtesy of friends who have also performed this operation, I am able to refer to their experiences in this summary. In general, their results agree fairly closely with mine. This report, therefore, summarizes the findings and results in 15 cases of ureteral denervation.

The shortcomings and inconsistencies of this clinical report are quite evident to us. We understand that in some cases one cannot tell positively whether the relief should be attributed to the ureteral denervation or some other operation which was performed at the same time. We are frank in stating that at the present time our opinions concerning ureteral denervation are in the formative stage. Nevertheless, we are quite unable to explain the remarkable cessation or diminution of discomfort that has occurred in most of these cases without assuming that the ureteral denervation had something to do with it.

We present this report, therefore, not as the final proof of a proposition, but merely to lay the proposition before you. In doing so, we hope that these observations on the innervation of the ureter and its behavior after denervation will convince us of the importance of further investigation of the genito-urinary organs from this viewpoint. After having spent a good deal of time in the last three or four years dissecting the nerve supply of the reproductive and urinary organs in human beings, I wonder whether our clinical concepts concerning many painful functional and pathological disturbances of these organs would not be greatly clarified and our therapeutic procedures better directed if we knew more about the innervation and the normal functional relationships of these structures.

THE ANATOMICAL BASIS FOR URETERAL DENERVATION

In the beginning, we did not have the assurance that a denervation of the ureter could be done without producing ureteral paralysis. Neither did we know that the ureter could be deprived of this extensive blood supply without resulting damage. Indeed, as late as 1931, it

*Chairman's Address, Section on Gynecology, Southern Medical Association, Twenty-Sixth Annual Meeting, Birmingham, Alabama, November 15-18, 1932.

*From the Department of Gynecology of the Johns Hopkins Hospital and University.

was stated that an operation such as we had planned and performed was not a denervation at all and that the ureter received no such nerve supply as we had hypothecated.

By clinical experience, pyelograms and functional tests, however, we have amply demonstrated that an operation such as we have been performing is safe, does not interfere with ureteral function and causes no early or late ureteral paralysis or dilatation. Our clinical experiences have since been confirmed by the admirable experimental work of Andler and Frommolt. In our own anatomical laboratories, on the basis of many dissections on human beings, we have also demonstrated that the ureter does receive an independent innervation between the bladder and kidney. Knowing this, we have been able to locate, excise and identify these nerves during ureteral denervations on patients.

We presented a summary of some of the anatomical and physiological problems in the *Journal of Urology*, in December, 1932. On the basis of this anatomical and physiological foundation, therefore, we are sure of our statement that the operation, as we perform it, is safe and will not harm the ureter. It remains to be seen whether it will do it any good.

OPERATIVE TECHNIC

The operation itself is so simple that it can be performd by any surgeon trained in gynecology or urology. Far more difficult than the operative technic is the careful diagnostic study and patient, conservative urological treatment and observation without which the operation should never be performed. We feel that, in a woman, the gynecological features should be considered just as carefully as are the urological.

In brief, the operation consists of a stripping of the ureter. It has been my custom to try to mobilize it completely, cutting all its lateral connections between the bladder and the third or fourth lumbar vertebra. In some of our cases, I have not been able to free the ureter completely to the bladder because of dense scar tissue in the broad ligament. We have noted this difficulty particularly in women who have had hysterectomy.

Since I see the problem of ureteral denervation from the viewpoint of a gynecologist, I have felt that in women this operation should usually be done transperitoneally where the lower ureter is involved. Some of my colleagues, however, have used the extraperitoneal route with excellent results. My reasons for advocating the abdominal route are two. In the first place, it seems to me that one can see and mobilize the lower ureter better through a midline abdominal incision than by an extraperitoneal approach. The chief advantage, however, is that it exposes not only the urinary tract, but also the reproductive organs and the whole intraperitoneal situation. In my series of 8 cases, for example, I found organic ovarian disease in two and pelvic and abdominal adhesions in several others. The transperitoneal route clinches the diagnosis completely, as far as the abdomen and pelvis are concerned. For these reasons, I usually employ it in women.

Assuming that the ureter has been exposed, one then strips it of all connections. While doing this, one will encounter at various levels, fine vascular bands, like delicate ribbons which pass to the ureter, chiefly from the median side. One can always find these if he separates the ureter gently from the retroperitoneal tissues. These bands are denser and more resistant than the areolar tissue and fat in which the ureter normally lies. If one saves these little bands of tissue and examines them microscopically, he will find that they consist chiefly of blood vessels, but that among the vessels are long nerves that pass directly to the ureter. We have located and identified these clusters of blood vessels and nerves in every case in the last three years. An idea of what they look like to the naked eye and microscopically may be gained from the dissections which we illustrate.

We take pains not to injure the adventitia of the ureter, because it contains a rich plexus of blood vessels and nerves. Also we have avoided the use of tension on the ureter. Except for cutting the peritoneum and these clusters of nerves and blood vessels, we perform most of the ureteral mobilization by blunt dissection. When the operation is done extraperitoneally, an indwelling ureteral catheter may assist in locating the ureter. At the conclusion of the operation, the ureter lies free just like a loose cord. After completing the dissection, the peritoneum is closed over the ureter by a continuous suture of fine catgut if one has performed the operation transperitoneally. Because of the rather extensive dissection that attends the extraperitoneal approach, it may be wise to drain the extraperitoneal wound in the iliac fossa. I have never drained the abdomen when the operation has been performed transperitoneally.

RESULTS

For a comprehensive view of the results, condensed charts are presented on pages 679 and 680.

Before attempting to analyse or explain the results, I shall summarize the present condition of the 8 women upon whom I operated. Two have been completely well and free from every suggestion of ureteral or renal pain since 1924 and 1927, respectively. Two are enjoying better health than they have had for many years and are no longer invalids in any sense. They occasionally have slight twinges in the side but these are inconsequential. Two others have had marked relief but are not completely well. They still have some pain at times, and are very nervous because of menopausal symptoms and one has some gastro-intestinal symptoms in the upper abdomen, for which we can find no explanation. In these 6 cases from one to nine years have passed since the ureteral denervation was done. The seventh patient of my own series, 25146, is a failure. My eighth case has been operated upon too recently to warrant any conclusions. It is perhaps worth relating that not one of these 8 women has required or received a cystoscopic treatment since ureteral denervation.

In addition to my own 8 cases, I know of seven that have been operated upon by other

CHART I.

Case	Diagnosis and Treatment Before Denervation	Final Operation	Result
20761 W. H. Female	Tbc. rt. kidney, nephrectomy, 1917. Cystitis, pyelitis, 1917-'20. Ureteral stricture, left; dilatations, 1919-'24. No hydronephrosis.	Denervation, left, 1924. Salpingect. Lt. oophorect. Sterilization.	Well
7701 U. M. H. Female	Hydronephrosis, bilateral, sterile. Ureteral stricture. Dilatations, 1922-1927.	Denervation, bilat. Jan. '27. May '27, hysterect., bilat. salpingoooph.	Well
19615 U. M. H. Female	Ureteral stricture, 1922. Hysterectomy, 1922. Dilatations, 1922-1929. Nephropexy, 1929. No hydronephrosis. Urine normal.	Denervation, right. Bilat. salpingoooph., Jan. 1930.	Almost no pain Menopausal symptoms
23357 U. M. H. Female	Nephropexy, 1928. Hysterectomy, 1929. Urine normal. Hydro-ureter. Stricture, dilatations, 1929-1931.	Denervation, right; right salpingoooph., 1931.	Practically well
23282 U. M. H. Female	Ureteral stricture, 1919. Suspension uterus, 1919. Dilatations, 1922-1931. Cystitis. No hydronephrosis.	Denervation, hysterectomy, left salpingoooph., 1932.	Greatly improved
25146 U. M. H. Female	Lt. ovary removed, 1907. Exploratory laparotomy, 1915. Lt. salpingectomy, 1921. Hysterectomy, 1923. Tonsillectomy, 1925. Stricture. 100 dilatations. No hydronephrosis.	Ureteral denervation, 1932	Failure
42795 J. H. H. Female	Nephroptosis; stricture, 50 dilatations. Nephropexy bilateral. Exploration rt. kidney. No hydronephrosis. Urine normal.	Ureteral denervation, 1932	Improved
Dr. S. Female	Nephralgia, left; nephropexy. Later nephrectomy. Stricture and dilatations, right ureter.	Ureteral denervation, extraperitoneal, 1931.	Improved
Dr. J. Male	Ruptured duodenal ulcer, 1926. Slight hydro-ureter. Renal ptosis. Ureter kinked, 1927.	Ureteral denervation, trans-peritoneal, 1927.	Well
Dr. H. Female	Nephroptosis, right. Pyelitis, right. Ureteral kinks, nephropexy, right, 1925. Temporary relief. Slight hydronephrosis.	Ureteral denervation, right, extraperitoneal, 1926.	Well

This chart shows the various urological and gynecological operations which have been performed in this series of 10 cases. With the exception of the sixth case, there is nothing unusual in these histories, for they represent the various methods by which urologists and gynecologists cooperate in the attack on genito-urinary problems in women. The only common and striking feature is the persistence of increasing ureteral pain in spite of all these procedures. The first 7 denervations in this series were done by me; the eighth by Dr. William Shallenberger of Atlanta, Georgia, the ninth by Dr. Edward Johnson of Baltimore and the tenth by Dr. Thomas Hepburn of Hartford, Connecticut. More recently cases have been done by Dr. Nathan Sears, Dr. Thomas S. Cullen, Dr. Guy L. Hunner, Dr. Thomas B. Sellers and myself.

surgeons. Two of these have been perfectly well since 1926 and 1927 respectively. The third, operated upon by Dr. Shallenberger in 1931, has been definitely better. I fear that the fourth, Dr. Sears' case, has shown no improvement since her operation eight months ago. The other three operations, by Dr. Cullen, Dr. Hunner and Dr. Sellers, have been performed too recently to warrant any deductions.

It can be seen that the results in these two series of cases agree fairly closely. This agreement is the more impressive since I have neither examined nor treated any of the patients who were operated upon by other men, and assisted in only one of the operations.

ANALYSIS OF CASES

In analysing these cases further, we find that they are of two types:

First, those in whom the urological disturbances are free from any extra-urinary disease;

Second, those in whom the urological situation is complicated by definite pathological conditions in the lower abdomen or pelvis, especially the reproductive organs.

Of my own 4 cases, one had had a nephrectomy for tuberculosis which was followed by pyelitis. This patient had received ureteral dilatations for five years and required five to eight grains of morphine daily during the worst attacks. The second had recurring attacks of cystitis and possibly pyelitis. In the third, the pain was attributed to nephroptosis but was not relieved by nephropexy, subsequent exploration of the kidney or fifty ureteral dilatations. The urologist in charge of this patient had suggested removing this painful kidney when I was called in consultation. The fourth had a very painful, spastic and tender ureter which felt like a lead pencil when palpated through the vaginal vault. She had no other genito-urinary disease. In all of these 4 women, the diagnosis of ureteral stricture had been made and they had received a total of approximately twenty-seven years of ureteral dilatations, one numbering over 100 treatments and another over 50. Of these

CHART 2.

Case	Reproductive Organs at Time of Denervation	Operation	Date	Result
20761 W. H.	Normal	Sterilization (bilat. salping.) Lt. oophorectomy. Lt. ureteral denervation	1924	Well
7701 U. M. H.	Looked normal. Lt. ovary later had chocolate cysts	(1) Bilateral denervation. (2) Hysterect. Bilat. salpingoooph.	Jan. 1927 May 1927	Well
19615 U. M. H.	Ovarian endometriosis	Bilat. salpingoooph. Ureteral denervation, right	1930	Almost no pain. Menopausal symptoms
22357 U. M. H.	Uterus, lt. tube and ovary missing. Rt. ovary cystic, adherent	Rt. salpingoooph. Rt. ureteral denervation	1931	No ureteral or renal pain. Slight discomfort in pelvis
23282 U. M. H.	Adhesions around lt. ovary and uterus	Lt. salpingoooph. Hysterectomy. Lt. ureteral denervation	Jan. 1932	No renal colic. No ureteral colic. Slight pelvic soreness
25146 U. M. H.	Uterus, lt. tube and left ovary missing. Rt. adnexae normal	Lt. ureteral denervation	May 1932	Less ureteral and renal colic Some pain deep in pelvis. Failure
42795 J. H. H.	Normal	Rt. ureteral denervation	June 1932	No sharp colic. Still has dull pain in right kidney and right ureter
Dr. Shallenberger's case	Missing	Rt. ureteral denervation, extraperitoneal	Nov. 1931	No sharp pain. Still some discomfort. No morphine
Dr. Johnson's case—male	Normal	Rt. ureteral denervation, transperitoneal	1927	Well
Dr. Hepburn's case	Not recorded	Rt. ureteral denervation, extraperitoneal	1926	Well

4 patients, the first 2 have been completely well since ureteral denervation in 1924 and 1927, the third has been vastly better and the fourth shows no significant improvement.

The situation in the other 6 cases of this group is somewhat similar. Dr. Hepburn first saw his patient in 1925, when she had been suffering from recurrent right renal colic and pyelitis. Cystoscopy showed a redundant and moderately dilated right ureter, pus cells from the right kidney, nephroptosis and sharp kink high at the uretero-pelvic junction with beginning hydronephrosis. The function of this kidney was normal. He did a fixation of the right kidney and mobilization of the uretero-pelvic junction in 1925 which gave complete relief for four months. Within a year the pain had become so severe again that morphine was frequently necessary. In 1926, he therefore did a ureteral denervation extraperitoneally, freed the ureter from the uretero-pelvic junction to a point below the iliac vessels. This was followed by complete and permanent relief. He obtained a pyelogram two years later, which showed distinct improvement, in that the ureter was not so dilated as it had been and the calyces were smaller and sharper, thus confirming our experience that the operation does not cause hydronephrosis or hydroureter. The pyelitis had disappeared.

Dr. Johnson's case was in a man upon whom he had operated for ruptured duodenal ulcer in 1926. Three months later this man developed sharp renal colic in the right side, frequency of urination with slight pyuria. Several cystoscopic examinations and treatments were given through the next months without relief. Pyelograms were essentially normal. In March, 1927, Dr. Johnson stripped the ureter transperitoneally from the uretero-pelvic junction down below the iliac vessels. This operation also resulted in complete and permanent cure.

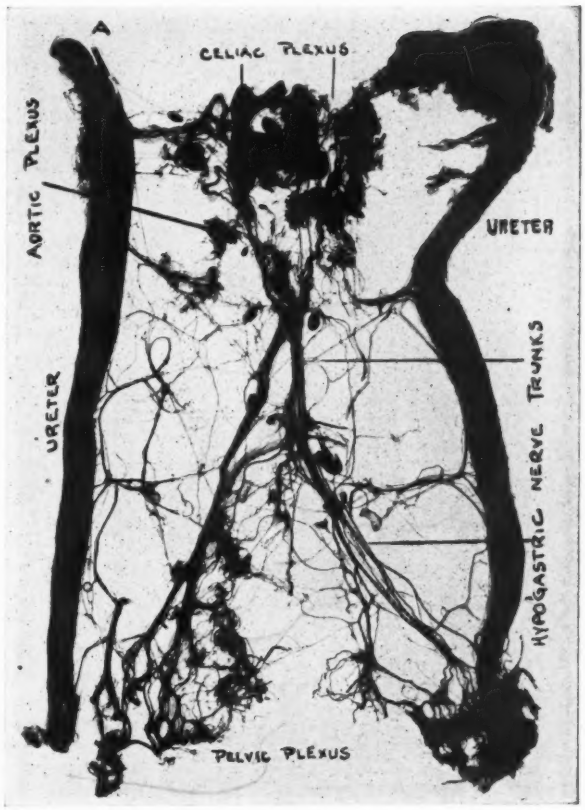

Fig. 1

Specimen 6472-2. Male fetus, age thirty-four weeks. An untouched photograph. The dissection shows both ureters, the celiac (solar) plexus which is continued downward as the aortic, hypogastric and pelvic nerve trunks and plexuses. The "presacral nerve" is shown to be in reality a large aggregation of nerve trunks. Numerous nerves pass to ureters from these abdominal sympathetic trunks, usually accompanied by blood vessels. There are practically no blood vessels in the central part of the dissection. Along the ureters, however, some of the finer blood vessels were spared as their removal might have damaged the accompanying nerves. (From the Journal of Urology, Dec., 1932.)

Dr. Shallenberger's experience is rather remarkable. As in my first case, he did a ureteral denervation on a woman who had only one kidney. His patient had had severe pain in the left kidney for years, probably following a difficult obstetrical experience. She had had two operations on her left kidney leaving a greatly damaged organ which had herniated through the wound in the flank. Repeated ureteral dilatations and conservative care failed to afford any relief. Finally, the left kidney was removed. Within eighteen months she began to have pain in her right kidney. Dr. Shallenberger found a stricture at the lower end of the right ureter and dilated it repeatedly without any benefit. She was also receiving large doses of morphia. In November, 1931, he therefore did a ureteral denervation extraperitoneally, stripping the lower ureter from the bladder up above the iliac vessels. This eliminated the sharp attacks completely, was followed by gain in weight and improvement in the general health. Dr. Shallenberger wrote to me in October 1932: "Although the patient continues to complain and has not been made entirely comfortab'e, she is of the type that probably never will be entirely well. But I am convinced that the operation has done a great deal for her and I would not hesitate to do the same thing again under similar circumstances."

Since Dr. Sellers performed his operation very recently his ultimate result is not known. The immediate result seems to be very good, however, for, according to a recent letter, this woman reported to Dr. Sellers that since this

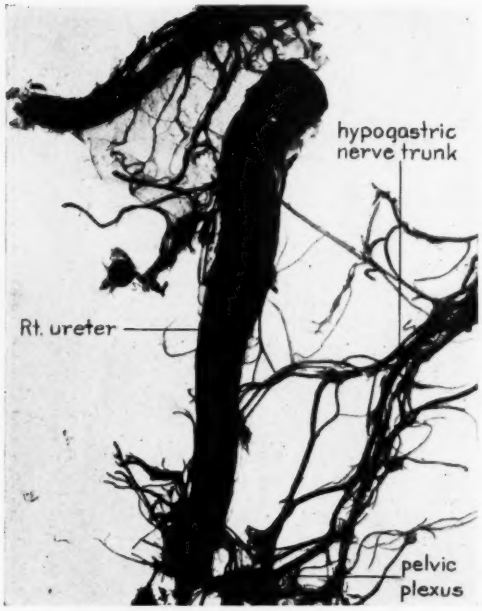

Fig. 2

Specimen 6304-2. Female fetus, one month before term. Siehler's stain. The specimen shows part of the right ureter, with the lower part of the hypogastric nerve trunks, part of the pelvic plexus, and the ovarian artery, vein and nerves. The juxtavesical part of the ureter is again seen to lie in the midst of a plexus of sympathetic nerves and ganglia. Some of these nerves surround the ureter just before it enters the bladder. In the upper part of the dissection one sees the ovarian plexus of artery, veins and nerves. In this one sees the definite connections between the ovarian or spermatic nerve plexus and the ureter. (From the Journal of Urology, Dec. 1932.)

operation she had felt relief for the first time in four years.

Dr. Hunner's case emphasizes one or two details which confirm our experiences.

In the first place the pyelo-ureterogram taken three weeks after the operation, showed no changes in the lumen of the ureter or kidney pelvis, thus again dispelling the old myth that such an operation would produce ureteral paralysis and dilatation. In the second place, he found that the passage of the wax bulb up this denervated ureter was distinctly less painful than it had been. Before the operation such procedures had invariably caused prolonged and severe ureteral colic, requiring repeated doses of morphine. After denervation, the patient demanded no sedative at all.

These postoperative studies confirm and amplify our experiences. We have pyelo-ureterograms obtained by three different urologists at intervals of three weeks, five months and two years after denervation, all showing that the operation that we have described did not produce postoperative ureteral stricture, dilatation or hydronephrosis. Two of us, moreover, have observed that ureteral denervation definitely lessens the sensitiveness of the ureter to catheterization.

As one can see, these cases are all entirely different. Some involve the upper ureter, others the lower; in some the operation was extraperitoneal, in others it was transperitoneal. In all of them the painful kidney and ureter were worth saving. Two showed a slight hydronephrosis, several had dilated ureters and ptosis and two had chronic pyelitis.

The failures are of course as important as the successes. Of these ten patients, two showed little or no benefit after ureteral denervation. I do not know the details of Dr. Sears' case. In my own several factors stand out.

This woman had received more than 100 ureteral dilatations, each of which had been followed by a severe reaction. She had been operated upon many times. We wonder just how much traumatism the human organism can stand. Since her denervation she has felt much less pain along the ureter and kidney, but now maintains that her worst discomfort has always been in the rectum and vagina and that this has not been affected by the ureteral denervation. Possibly we overlooked this complaint, being overwhelmed by her long urological history. At any rate, I confess that I do not know what is wrong with this woman.

Ureteral or Renal Pain.—In these cases, we find that one of the problems has been to determine whether the ureter or the kidney has been the source of pain. This is reflected in the fact that four nephropexies had been done and one exploration of the kidney. Most of these operations gave temporary relief just as ureteral dilatations had. In these patients, however, it seems that nephropexy was not the answer to the problem, although we all know that in indicated cases it affords remarkable relief.

It is not our object to discuss renal fixation in this article, except to state that it is important to distinguish the indication for nephropexy and ureteral denervation. This distinction may be difficult. Indeed, a patient may need both operations and one urologist did both a nephropexy and a ureteral denervation at the same time with results that seem to be promising.

These records also show that it may be a serious matter to remove a fairly normal kidney for pain. In this series, there were fortunately none who had had nephrectomy for pain alone, although 2 had only one kidney and a third was on the point of entering a hospital to have

a normal kidney removed wholly because of pain. This woman has been almost perfectly well since ureteral denervation which was done instead of nephrectomy. On the whole, we find it hard to justify the removal of a functioning kidney for pain, especially in a young and healthy person. It should never be done, at least, without the consideration of every possible alternative course.

In summary, we present 10 cases of ureteral denervation in which there were no complicating extra-urological problems. The ureteral denervations were done by myself (4 cases) and six other surgeons. In practically all of these cases, the decision to do a ureteral denervation was reached after years of dilatations had resulted in failure. In one of my cases, the ureteral denervation saved a normal kidney which was about to be removed because of pain. Of these 10 operations, 3 were done too recently to warrant any conclusions. Of the remaining 7, 2 resulted in failure, and 5 in complete cure or definite improvement. There has been no postoperative hydro-ureter or hydronephrosis and the operation has not impaired renal function.

COMPLICATED UROLOGICAL CASES

In this second group are 5 cases, 4 of which were operated upon by myself and one by Dr. Cullen. In this type we face a complicated problem in that the outstanding pain is ureteral and gross pathological changes are found in the uterus, tubes, ovaries and possibly the appendix. In addition one may find organic changes in the urinary organs, such as hydro-ureter, hydronephrosis or chronic pyelitis. Under these circumstances, then, our problem is to study the relationships and significance of these two groups of disorders, and determine if possible, whether the urinary disorder is an independent affair or whether it is entirely secondary to or influenced by the pathological processes in the uterus, tubes or ovaries.

We might summarize the results of this group of 5 cases before analysing the clinical problems. Two of these oprations (Wharton and Cullen) were done too recently to warrant conclusions. Of the other 3, 2 have been perfectly well since 1927 and 1931, and the third has very little discomfort in the region of the ureter or kidney. Although her operation was done in 1930, she still is disturbed by menopausal symptoms and discomforts which vary in location all over the body. The menopausal symptoms came on after the removal of the ovaries and denervation of the ureter.

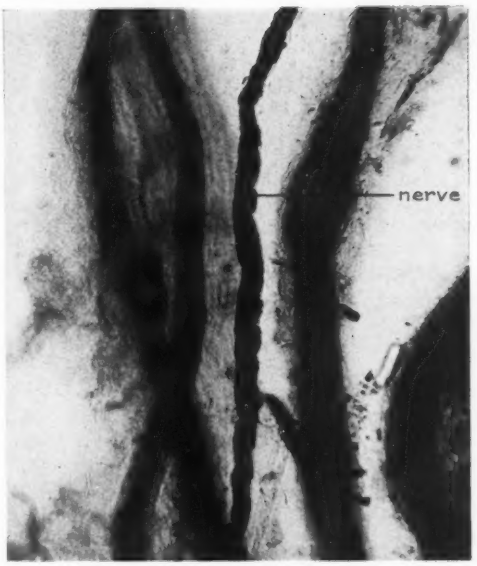

Fig. 3

Section 5916. The Union Memorial Hospital. Siehler's stain. One could hardly mistake the identification of the nerve, one of several removed during a denervation for ureteral colic. (From the Journal of Urology, Dec. 1932.)

In these cases, the fundamental requisite is the correct diagnosis and, unfortunately, this is not always easily obtained. The usual difficulties that may be present in any simple genito-urinary case are magnified here because both systems are involved. As a consequence, patients of this sort tax to the utmost our resources of diagnostic skill, patience and judgment. It is this type of patient that has suffered almost endlessly from interminable ureteral dilatations and gynecological operations.

The ovary has perhaps caused more confusion than any other organ in the pelvis. Nor is this remarkable in view of the frequency with which uncomplicated ovarian disease such as endometriosis, chocolate cyst and early carcinoma are either missed entirely or mistaken for salpingitis, myomata uteri or chronic appendicitis. When such an error in diagnosis involves only the gynecological organs, it should not be a serious matter for it is corrected when the abdomen is opened, and does not interfere with the immediate institution of proper surgical treatment. When we add the ureter to these responsibilities, however, a new situation is created, for the ureter is an extraperitoneal organ about which an abdominal exploratory gives us no information whatever.

Fig. 4

Part of the tissue from case, Surgical No. 90043. The Johns Hopkins Hospital. Siehler's stain X400. The end of a nerve fiber is seen here showing the "brush-broom" effect produced by the separation of the individual fibers when a nerve is cut. (From the Journal of Urology, Dec. 1932.)

The clinical intimacy between the ovary and ureter is readily explained on the basis of their anatomical propinquity, their embryological consanguinity, and, as we showed in 1932, their intermingled innervation. In addition to this, the ovary has confusing habits. Today it may be of normal size, and in three weeks it may contain a cyst as large as an orange, which may again disappear in a short time. Then, again, I am quite convinced in my own mind that an ovary may be definitely responsible for pain even though it looks normal. I could record the details of three or four cases in which I have fairly good evidence that long before the ovary showed gross pathological changes, it was definitely the cause of pain, and thereby produced endless confusion. In these cases, the endometrial rests or cystic changes seem to be buried deeply within the ovarian stroma, entirely concealed and impalpable.

When one finds definite gynecological disease, usually it has seemed wise to restore the pelvis to a healthy state before undertaking an extensive course of urological treatment, because the gynecological operation can be done easily and decisively and one would hardly expect to obtain a state of genito-urinary well being with palpably diseased reproductive organs. If the pain persists after the gynecological operation, one can then attack the urological problem knowing that the gynecological situation is clear.

There is, however, another way to handle such situations which may be more satisfactory at times. In some of these cases, it may be safe or advisable to postpone the gynecological operation and try to control the ureteral discomfort by conservative measures with the clear understanding that a gynecological operation may be necessary. If this course brings relief, one may be able to postpone the gynecological operation for a long time. On the other hand, if the pain persists, one may later be in a position to perform both the ureteral denervation and the gynecological operation at the same time. This latter course eliminates one operation. It also enables the surgeon to get better acquainted with the situation and to estimate the significance of the genito-urinary problem. This course can be followed, however, only when the gynecological situation can be postponed with safety and when there is mutual confidence and understanding between the patient and the physician. As our summary shows, we have performed denervation of the ureter and gynecological operations simultaneously on several of these women with satisfactory results.

Whichever course is followed, it is fundamentally necessary that both the surgical and urological treatment should be done carefully, conservatively and thoroughly. After the gynecological operation, it should usually be possible to dismiss the pelvis as a source of pain. We can understand the interminable ureteral treatments that some of these patients received before any other course was available but it is hard to explain the necessity of a dozen major operations to correct disorders of the reproductive organs.

POSTOPERATIVE CONVALESCENCE

None of my patients has developed any urinary complication after ureteral denervation. The usual number have required catheterization of the bladder for a day or two but there have been no instances of any serious surgical or urological complications. This is, however, a major operation on a vital part of the urinary system and accidents will undoubtedly occur. We hope that they will be reported to us. We feel that the point of greatest danger lies in the way the ureter is handled. We have consistently recommended that the ureter should be traumatized as little as possible either by traction on tapes or surgical manipulation. We have always advised against injuring the adventitia because it carries a rich plexus of

blood vessels and nerves. It is necessary to understand clearly that the denervation consists of cutting the nerves that go to the ureter with the least possible manipulation of the ureter itself. If one injures the adventitia or the ureteral wall, one can hardly expect anything but scar tissue and possibly stricture as a consequence.

In addition to opening up the entire question of the origin of ureteral peristalsis, the histology of the neuromuscular mechanism in the ureter and the existence of a sensory nerve supply, ureteral denervation raises some clinical questions which we cannot explain. One of them has to do with the time required for the disappearance of ureteral spasm and pain after denervation. Occasionally the ureteral pain disappears immediately after the operation and never returns. That is what one would expect if the sensory nerve supply of the ureter were severed completely. At times, however, the pain does not disappear in this abrupt manner but may be felt occasionally for weeks or months afterwards. In one such case it ceased completely and permanently after four months and in the other it has always been quite insignificant. It is therefore impossible to tell what the ultimate result will be until at least several months have elapsed.

HOW LONG SHOULD ONE CONTINUE URETERAL DILATATIONS?

In general we feel that in two or three years one should be able to estimate with fair accuracy whether an individual will react favorably to conservative measures. In some of the patients under discussion ureteral denervation was done after less than a year of observation and treatment, and excellent results were obtained. We see no harm in operating as soon as one is convinced that the painful ureteralgia will not yield otherwise. On the contrary, we feel that undue ureteral traumatism too often repeated and carried on for many years, creates a condition which militates against any therapy. The worst result that we have seen was in a woman who had suffered more than 100 ureteral dilatations.

Consequently we should be inclined to suggest ureteral denervation as soon as we are convinced that other measures are futile, and our decision to intervene surgically is hastened if we suspect that there is some pathological process in the reproductive organs or the appendix which maintains ill health or produces ureteral irritation. One should, of course, study every case with infinite pains and be certain that ureteral denervation is the operation which is needed.

In handling these trying cases, it seems to me that one of the fundamental requisites is patience, supported by conservatism and thoroughness. This mental background is obtainable only, of course, by virtue of painstaking urological and gynecological study. As Dr. Quinby remarked in discussing Dr. Hepburn's case before the New England Surgical Society, "These individuals demand from their medical consultant a degree of patience and endurance much beyond the ordinary." Their resistance having been shattered by unrelenting pain, fruitless urological and surgical procedures, they cannot be treated as one would handle an average person. It is only by putting one's self in their place that one can appreciate their viewpoint and avoid being irritated by them. In this respect, if ureteral denervation should prove successful, it might afford almost as much relief to the doctors as to the patients themselves.

In conclusion we are presenting a series of 15 cases of ureteral denervation, done by myself and seven other surgeons. This is the first comprehensive series of ureteral denervations that has been published. In accumulating this information, it is not our object to try to prove any proposition whatever. Although we are encouraged by the results, we still regard this new operation as having many of the features of a surgical experiment. We trust that it will be used conservatively.

SUMMARY

(1) The clinical features of a series of 15 cases of ureteral denervation are summarized.

(2) The operative technic is described, and reference is made to the anatomical and physiological data on which the operation is based. It is our experience that the operation which we have performed does not in any way impair the function of the urinary organs. Reference is made to experimental work which confirms this experience.

(3) The diagnostic problems have been presented. They involve a careful study of the patient from every viewpoint, with especial emphasis on those organs which may produce similar syndromes or which may perpetuate ureteral disturbances. The ovary has been one of the chief stumbling blocks to a correct diagnosis. A correct diagnosis at the outset may save several years of unnecessary expense and discomfort. In many cases the differential diag-

nosis is extremely difficult, and in some instances impossible until the pathological picture is evident. This emphasizes the necessity for conservatism.

(4) The results of ureteral denervation have been gratifying, not only in our hands, but also in the experience of other surgeons. Of a total of 15 cases, (8 my own, 7 operated upon by others), 4 are perfectly well, 2 are practically free from pain, 3 definitely better, 2 are failures, and in 4 the operations were done too recently to warrant conclusions. Some rather interesting observations have been recorded concerning the reactions of these patients after ureteral denervation.

(5) In some of these cases, it is clear that ureteral denervation has solved a very distressing problem. The operation must be regarded as having many of the features of a surgical experiment. If it has any inherent value, its successful utilization will depend largely upon the accuracy of the diagnosis, the thoroughness with which each case is studied and the care with which the operation is performed.*

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PULMONARY ABSCESS IN CHILDREN*

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Pulmonary abscess in children has been reported with increasing frequency during the past decade. Among 20,000 children admitted to a large children's hospital, 31 had lung abscess and 2 gangrene of the lung, a total of 33 cases.[1] Among 2,255 cases of lung abscess collected from the American literature, mostly from clinics for adults in which children rarely were admitted, 59 were infants and children.[2 3 4 5 6 7] Among 700 cases admitted to the Pediatric Service at the Duke Hospital were 6 cases of lung abscess.

The figures of Flick and his co-workers[7] suggest that the frequency of this condition increases with age from infancy to middle life, and then declines, for among 172 cases 6.4 per cent were in the first decade, 10.5 per cent in the second, 27.9 per cent in the third, 30.8 per cent in the fourth, 18.6 per cent in the fifth, 3.6 per cent in the sixth, and 2.3 per cent in the seventh decade of life. In adults approximately three times as many males as females have pulmonary abscess. Of the six children reported in this paper, five were males and one female. They varied in age from one and one-half to thirteen years.

The predisposing causes of abscess in 39 cases[2 3 4 5 6 7] were: pneumonia in 22, tonsillectomy in 10, aspiration of a foreign body in 5, exposure in 1, and aspiration of infected material from the pharynx in one case of Vincent's angina.

Pulmonary abscesses can be divided on the basis of their etiology into: (1) bacterial ab-

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scesses, for which there is no specific treatment other than vaccines; (2) fuso-spirochetal abscesses, which require arsenical therapy; and (3) the mycotic or fungus infections for which iodides are necessary. All of these patients require general medical care, and surgical operations when necessary, regardless of the type of abscess or the nature of specific treatment. The prognosis usually is hopeless if there are multiple embolic pulmonary abscesses following septicemia or pyemia. The majority of these infections in children, as in adults, however, are single lesions resulting from aspiration of infected material from the mouth. They may be absorbed, may discharge through a bronchus, or may be drained surgically through the chest wall.

BACTERIAL ABSCESS

Lung abscesses due to bacterial infections are relatively more important in children than in adults. Staphylococci are the most common infecting organisms, although Friedlander's bacillus, *B. coli*, streptococci, *B. pyocyaneus*, *B. influenzae*, and organisms of the gas gangrene group also may be responsible. *Case 1* is an example of a staphylococcus pulmonary abscess complicated by rupture of the abscess into the pleural cavity and consequent pyo-pneumothorax. The patient recovered after the pleura was drained by Hart's tidal-irrigation method.[8]

Case 1.—J. S., No. 2276, a white boy, 2 years of age, had symptoms of acute pneumonia on December 3, 1930, became steadily worse, and was admitted to the Duke Hospital on December 9, 1930. The patient was pale and cyanotic and, though conscious, was too ill to cry. The temperature was 40.2° C., the pulse 150, and the respirations 72. The white blood count showed 24,000 cells. The upper respiratory tract was not inflamed. The percussion note over the right chest was dull, except for resonance at the apex. Medium fine rales were heard over the entire lung, although the breath sounds were normal. The roentgenogram demonstrated a dense process involving the entire right lung, except the apex and the extreme base, a small area of pneumothorax along the lateral chest wall, and a cavity in the middle of the right lung.

A transfusion, given the day of admission, caused marked improvement in the child's condition. Four days later signs of pneumothorax were found and confirmed by x-ray. There was marked displacement of the heart accompanied by respiratory distress. Thoracentesis revealed a positive pressure and a small amount of pus. Following the removal of the air, the child's condition was greatly improved. *Staphylococcus aureus* was grown from the pus. The following day signs of fluid were noted and a large amount of pus was found. An inter-rib puncture was done and tidal drainage was instituted, with dramatic symptomatic improvement. Blood cultures made seven days after admission contained 6 to 7 co'onies per c. c. of *Staphylococcus aureus*. The patient's condition gradually improved, following blood transfusions on the ninth and fourteenth days, so that on December 30, 1930, three weeks after admission, the temperature became normal. The roentgenogram at this time showed the right lung to be two-thirds expanded with no evidence of fluid in the pleural cavity. Ten days later the lungs were

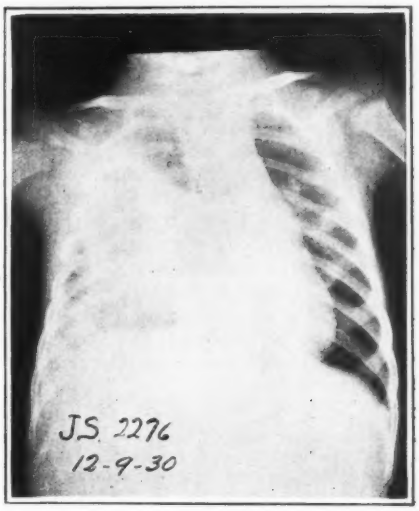

Fig. 1, Case 1
Roentgenogram on December 9, 1930, showing staphylococcus abscess of lung, with pneumothorax and displacement of the mediastinal structures.

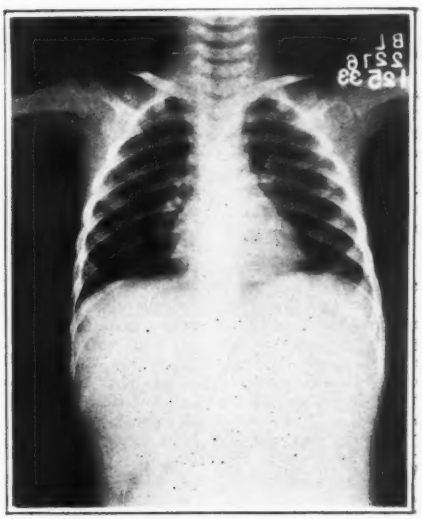

Fig. 2, Case 1
Roentgenogram January 5, 1933, three years after illness, showing normal lung fields.

much clearer, but there was marked pleural thickening along the lateral chest wall. No evidence of a cavity remained. The child when seen on January 25, 1933, more than two years after his illness, was normal to physical examination, and the roentgenogram revealed no lesion of the lungs.

FUSOSPIROCHETAL PULMONARY ABSCESS

One of the more common types of pulmonary abscess in children is caused by fusospirochetal organisms, which are present in infected tonsils, gingivitis, and in Vincent's angina. These organisms, obtained around the teeth, will produce typical pulmonary abscesses and gangrene when introduced into the lungs of experimental animals by aspiration.[9] The lesion produced is due to a true symbiosis of organisms, for spirochetes, fusiform bacilli, vibrios or cocci introduced alone in pure cultures into animals will not produce abscess while pure cultures of the four, mixed and injected together, cause the typical lesion.[10] In *Case 2* the patient may have aspirated the fusospirochetal organisms directly from his infected tonsils and gums.

Case 2.—J. H., No. 2382, a white boy, 11 years of age, had otitis media for six weeks during August and September, 1930, followed by acute arthritis in both knees for one week. Both knees, however, remained stiff and were painful on motion. He was admitted to the Duke Hospital on January 14, 1931, when his temperature was 38.6° C. He had considerable dental caries, with an abscess of the first upper molar on the left side; small and scarred tonsils with evidence of chronic infection; and slightly swollen and tender knees, flexed at angles of about 90°, giving intense pain on motion. His chest was normal. Hemolytic streptococci were grown from the tonsils. On January 20, 1931, after the temperature had been normal for six days, a general anesthetic was given, the tonsils and adenoids were removed, and both knees were manipulated, the right one being placed in a cast. The abscessed tooth was broken in an attempt to extract it. Following these procedures the temperature rose and remained between 38 and 40.5° C. continuously. Nine days after the operation 180 c. c. of pus, containing hemolytic streptococci, were aspirated from the left knee. Three days later the patient had pain in the left chest, difficulty in respiration, and blood streaked sputum. Dullness, bronchial breathing, and many fine rales were found over the left lower lobe. Death occurred within 24 hours after the development of the respiratory symptoms. Just before death a diffuse pneumonia of both lungs, especially of the left, and a large area of cavitation in the mid-portion of each lung were demonstrated by roentgenogram. Bilateral fibrino-purulent pleurisy with a large amount of foul smelling pus, and single cavities in the left and right lower lobes, with ragged green and necrotic walls, were found at necropsy. These cavities contained foul green pus in which enormous numbers of fusiform bacilli and spirochetes were present. Both knees showed purulent arthritis.

In *Case 3*, the diagnosis of post-tonsillectomy pulmonary abscess of the fusospirochetal type was made within ten days after operation, arsenical treatment was started at once, and the patient promptly recovered. Two other patients, *Cases 4* and *5*, also received treatment, but evidently too late in the course of the disease, for little improvement was noted. Kline and Berger[3] have shown that 11 of 15 adults recovered following the administration of neoarsphenamine or sulpharsphenamine during the acute stage of this type of abscess. Smith[11] has reported the recovery of 13 out of 15 patients suffering from fusospirochetal lung abscesses in whom treatment was begun before the end of the second week.

Case 3.—J. P., No. 14180, a colored boy, seven years of age, had a severe cold with bronchitis for two weeks before a tonsillectomy, which was performed on May 6, 1932, while the infection was still present. After the operation the child had a daily elevation of temperature accompanied by productive cough, which became more severe. Eight days later abundant blood streaked sputum was raised. Ten days after the tonsillectomy, when he was admitted to the Duke Hospital, his throat was slightly inflamed and he had moderate enlargement of the posterior cervical lymph nodes. The temperature was 39.6° C., the pulse 124, and the respirations 36. There were definite signs of consolidation in the scapular and axillary regions of the right lung with impairment of the percussion note, bronchial breathing, and many fine rales. The roentgenogram showed a dense shadow in this area. Sputum examinations revealed large numbers of fusiform bacilli and spirochetes, and a few colonies of staphylococci and hemolytic streptococci grew in cultures. The white

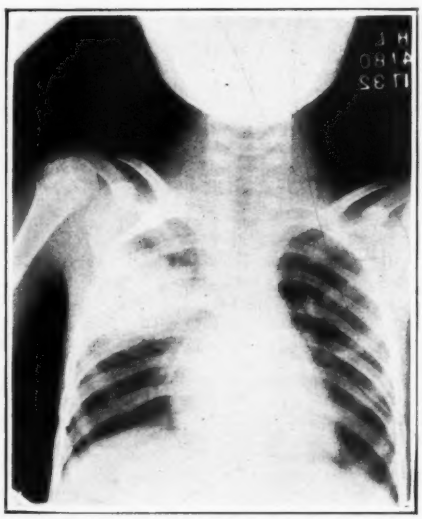

Fig 3, Case 3
Roentgenogram on May 17, 1932, eleven days after tonsillectomy, showing early fusospirochetal abscess.

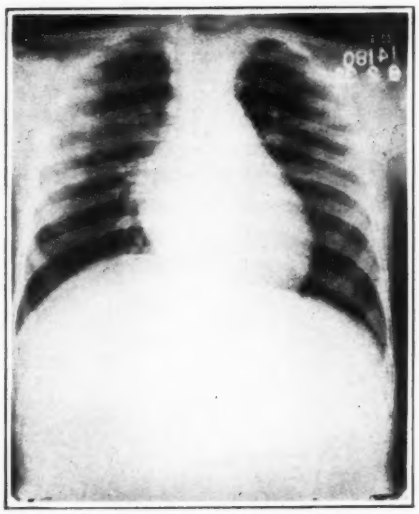

Fig. 4, Case 3
Roentgenogram on June 2, 1932, two weeks after Fig. 3, showing complete disappearance of lesion.

blood count was 25,000, with 92 per cent polymorphonuclear neutrophils. The tuberculin test (0.1 mg.) and the blood Wassermann reaction were negative. A diagnosis of early fusospirochetal abscess was made and the child received four injections of sulpharsphenamine (0.2 gm.) on alternate days. The amount of sputum gradually decreased and the temperature became normal after six days. Roentgenograms showed progressive clearing of the lesion, which disappeared sixteen days after admission. He was discharged, apparently well, four days later.

Case 4.—J. B., No. 7708, a white boy, aged 11 years, who had previously been well, became weak and listless after tonsillectomy in May, 1931. A few weeks later a productive cough appeared. On July 11, 1931, he consulted a physician who found a temperature of 38.8° C., a cough productive of a muco-purulent sputum, and diminished resonance with distant breath sounds over the right base. The patient's symptoms continued, pain developed in the right chest on September 10, 1931, and five days later, four months after the operation, he was admitted to the Duke Hospital. The patient was thin, chronically ill, and coughed up large quantities of foul, muco-purulent sputum. His temperature was 38.3° C. There was dullness over the base of the right lung extending into the right axillary region. Breath sounds were distant and vocal fremitus was diminished in this area. Darkfield examination of the sputum revealed large numbers of fusiform bacilli and spirochetes. There was a slight secondary anemia and a leucocytosis of 14,200 cells, with 76 per cent polymorphonuclear neutrophils. The roentgenogram showed an abscess cavity in the right middle lobe near the bronchus, with diffuse infiltration below this area, probably secondary to the abscess. The right diaphragm was pulled up by adhesions, and the pleura over the right lung was thickened. A diagnosis of fusospirochetal abscess of the lung was made and, the day after admission, 0.3 gm. of sulpharsphenamine was given intravenously, and repeated twice at intervals of four days. Two weeks later the roentgenogram showed little change in the appearance of the lesion and the patient went home against advice. When seen in the dispensary, three weeks after discharge, another abscess cavity was found about 2 cm. below the original one, and there was a large amount of infiltration throughout the entire right lower lobe. The patient refused to enter the hospital and has not been heard from since that time.

Case 5.—O. K., No. 13111, a white boy, 10 years of age, had a series of daily chills for two weeks during August, 1931, followed by a productive cough. The sputum at first was mucopurulent, but five months later became blood streaked and finally contained clots of blood. He was admitted to another hospital and a diagnosis of tuberculosis was considered. After a pneumothorax had been induced in the right chest the child became acutely ill, and a collection of purulent fluid was found in the right pleural cavity. When admitted to the Duke Hospital on April 10, 1932, the temperature was 39.0° C., the pulse 112, and the respirations 44. He was in obvious respiratory distress and in a very weak condition. There was dullness over the entire right chest with flatness laterally. Breath sounds were absent at the base and diminished elsewhere, while fine and coarse rales were numerous over the mid-portion of the right lung. The heart was slightly displaced to the left; the liver was 6 cm. below the costal margin and was soft but not tender. The roentgenogram showed marked density of the right lung with occasional small pockets of pneumothorax. There was a slight secondary anemia and a leucocytosis of 28,000 cells. The intradermal tuberculin test (0.1 mg.) was negative. A large amount of foul pus was located, by aspiration, in the right pleural cavity, whereupon an inter-rib cannula was inserted and closed drainage was instituted (Hart's method). The pus obtained from the pleural cavity contained short chains of gram-positive cocci and diplococci, but cultures were sterile. For three months after admission there was little change in the condition of the patient. On three occasions a few fusiform bacilli and spirochetes were found in the sputum. Large amounts of foul pus were discharged from the right chest and similar material, occasionally blood streaked, was coughed up. Cultures for fungi were negative. Five blood transfusions and nine intravenous injections of neoarsphenamine (0.2 gm. each) were given. Two ribs were resected (July 15, 1932) and an opening was made into the pleural cavity. A large abscess was found in the right lower lobe of the lung, and in addition, a very small empyema cavity. The surrounding lung tissue was necrotic. Following this operation drainage from the chest was copious and the child's condition improved markedly. After a few days his temperature reached normal for the first time, and remained below 37.2° C. until the time of discharge on September 18, 1932, six months after admission, and two months after the operation. The roentgenogram at this time showed the right lung to be quite dense with very little change in its appearance. The wound in the chest wall healed three months later. The child was seen February 1, 1933, after an interval of five months, during which time he had gained 3 kilograms in weight. He appeared much improved, but the roentgenogram showed little change in the appearance of the right lung.

MYCOTIC ABSCESS

The third type of abscess in children is caused by any one of a number of different types of fungi; Aspergillus, Monilia, *Torula hystolytica*, Oidium, Blastomyces, *Coccidioides immitis*, Sporothrix, *Streptothrix mucor*, and Actinomyces. Patients with these infections should be treated intensively with potassium iodide by mouth and ethyl iodide by inhalation. *Case 6* is an example of actinomycotic infection of the lungs.

Case 6.—G. B., No. 667, a white girl, aged 8 years, had an oat head caught in her throat in June, 1928. In November of that year she had a large swelling in the left side of her neck which was opened a month later, forming a chronic sinus. A few weeks later two sinuses opened spontaneously in the neck. From January, 1929, until September, 1930, new abscesses continually appeared, were opened, and drained chronically, until in September, 1930, she had draining sinuses in both axillae, three on the upper back, four on the chest, three on the lower back and two in the lower abdomen. One abscess had ruptured and was draining through the rectum. There was progressive loss of weight, loss of appetite, fretfulness, chronic cough and low grade fever. An abscess in the right hip region had produced a flexion deformity. On her first visit to the Duke Hospital, September 5, 1930, two years after the onset of her disease, the patient was extremely emaciated and pale, and had marked deformity of the chest, with sinuses anteriorly, posteriorly, and in the axilla, several of which whistled when she breathed. There were draining sinuses in the lower back, in the lower part of the abdomen, and in the right hip region. The discharge was thick yellow pus containing dark orange granules. A flexion deformity of the right hip and knee, marked scoliosis of the spine, clubbing of the fingers and toes, muscular atrophy, and a marked secondary anemia were present. Roentgenograms of the chest showed cavities in both upper lobes, with a diffuse infiltration throughout the lungs. The blood Wassermann reaction and tuberculin tests (0.1 and 1.0 mg.) were negative. Branching forms of actinomycetes were seen in smears of the pus from the sinuses, while cultures revealed staphylococci. Potassium iodide by mouth and ethyl iodide by inhalation have been given from September, 1930, until the present time. Orthopedic measures were used to correct the contractures of the right leg. During the first year of treatment many of the sinuses continued to discharge and new ones appeared, but by the end of the second year only two sinuses remained, and the child had improved markedly. She is now able to be fairly active about the house. However, marked deformities of the chest, due to fibrosis of the thoracic lesions, have developed. Roentgenograms on August 1, 1932, showed the lower lobes to be clear, but the left upper lobe was distorted, and the cavity was larger. That in the right upper lobe had decreased in size, but a second cavity had appeared, filling the apex.

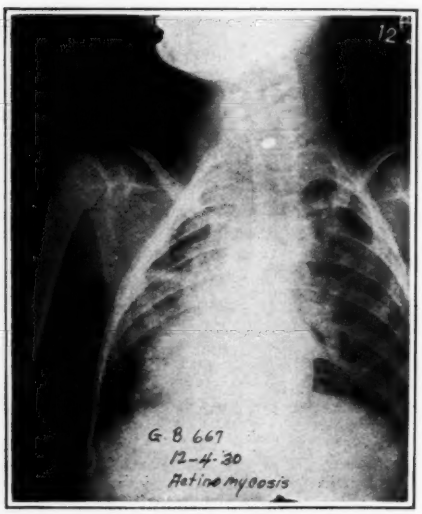

Fig. 5, Case 6
Roentgenogram of chest December 4, 1930, two years after onset of actinomycotic infection, showing extensive bilateral fibrosis and cavitation. Patient doing well, steadily improving five years after onset.

DIAGNOSIS

The history, physical signs, and x-ray shadows of bacterial abscess, mycotic abscess, and fusospirochetal abscess may be almost identical, and it is very difficult at times to differentiate any one of the three types of abscess, in the initial stage, from primary tuberculosis. The final diagnosis must rest upon a study of the sputum, which should be cultured on blood agar under aerobic and anaerobic conditions to demonstrate or eliminate the bacterial type of abscess. The sputum should be cultured aerobically on Sabouraud's media for the yeast-like and mould-like fungi, and anaerobically on blood agar for the Actinomyces and Streptothrix groups of fungi. The sputum, and the pus from discharging sinuses, should be examined for sulphur granules. Gram stains should be used in searching for Actinomyces and Streptothrix organisms. Fresh sputum should be studied under the darkfield for spirochetes, and it should be stained with gentian violet to determine the presence or the absence of fusiform bacilli. Many patients suffering from fusospirochetal abscess, who recover with arsenical treatment, have sputum from which staphylococci, pneumococci, and streptococci can be cultured on aerobic plates. In many patients suffering from lung abscess, especially very young children, sputum is difficult to obtain until late in the disease. Therefore, the history and the clinical observations are most important. Although both bacterial and mycotic diseases may follow operations, the fusospirochetal type is by far the most common postoperative infec-

tion of the lungs. The early treatment of these patients is so necessary that every patient who has fever, and physical or roentgenological signs of pulmonary infection after an operation, should be given a small dose of sulpharsphenamine or neoarsphenamine as soon as possible. We have seen two small children who died of postpneumonic abscesses of the fusospirochetal type, to whom arsenic was not given for three or four weeks after the onset, because no sputum was obtained and fusospirochetal organisms were not demonstrated. Both of these patients had definite gingivitis with fusospirochetal organisms about the gums. At present every child who has any pulmonary disease and oral sepsis associated with fusospirochetal organisms is treated with arsenicals if the presence of tuberculosis has been eliminated.

TREATMENT

The bacterial type of pulmonary abscess should be treated palliatively unless rupture into the pleural cavity and empyema occur. There is no specific therapy other than vaccines for this group of patients.

The fusospirochetal type of abscess must be diagnosed within the first two weeks of the disease if arsenical therapy is to be efficient in its cure. The more chronic infections should also be treated with arsenicals prior to surgical drainage. Because of the danger of causing rupture of the abscess into the pleural cavity, a therapeutic pneumothorax should not be induced.

Mycotic abscess of the lung should be treated by gradually increasing doses of potassium iodide. Patients suffering from resistant infections should receive, in addition, inhalations of ethyl iodide, beginning with 0.5 c. c. to 1.0 c. c. once daily, gradually increasing the dosage to 2 c. c. three times daily.

SUMMARY

Six cases of pulmonary abscess in children are reported, one due to bacteria, 4 to fusospirochetal organisms, and one to Actinomyces. The differential diagnosis and the specific treatment are discussed.

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DISCUSSION (*Abstract*)

Dr. W. Ambrose McGee, Richmond, Va.—There is hardly an article in the literature on this subject that does not refer to Dr. Smith's experimental and clinical studies of lung abscess. The literature on lung abscess leaves one with a great deal of confusion regarding the mode of production, its exciting cause and the treatment.

While Dr. Smith and many others, as Richardson, Lemon and Moore, have produced experimental lung abscess in different animals by aspiratory methods, many others, as Cutler, Fetterolf and Fox, and Schlueter and Weidlen, maintain that lung abscess is formed through emboli. Each group has good evidence on its side. Hunter could produce an aspiratory abscess only after previously traumatizing the bronchial mucous membrane. Cutler feels that immunity is another factor in the formation of abscess. Those in favor of the embolic theory feel that the large percentage of cases of lung abscess secondary to tonsillectomy is due to thrombi let loose in the blood stream by manipulation of the tonsils.

Besides the controversy regarding the production of lung abscess, there is one pertaining to the exciting cause. Rona and Castellani were the first to call attention to spirochetes in relation to broncho-pulmonary lesions. In 1923, Kline suggested this relationship and in the past few years Dr. Smith has reported the finding of fusospirochetal organisms in 90 per cent of the cases studied. If those anaerobic bacteria were isolated in the early stages of lung abscess, he was able to cure the condition rapidly by use of arsphenamine.

In a careful study of anaerobic bacteria obtained from lung abscesses, Cohn found *B. fusiform* in 6 cases, vibrios in 1 case, but *B. melaninogenicum* and streptococcus in 14 cases. The latter two organisms seem to present another example of symbiosis and possibly raise the question as to whether or not that combination has a bearing on the etiology of lung abscess.

A diagnosis of lung abscess in the early stages is most difficult. Such symptoms as persistent cough, chest pain, toxemia, slight or marked fever and tachycardia with no dyspnea following a protracted case of pneumonia or an operation in a possible infected area, suggest a lung abscess. Kerley feels that an aspiratory lung abscess can be differentiated from an embolic one. In the former the lesion is single, is practically always in the right lung, is seldom near the periphery but tends to develop near the hilum, is not sharply defined in the early stage and at first appears as a vague ill-defined opacity which becomes denser and sharper in outline as it becomes chronic. Embolic ones may develop anywhere in either lung, are usually near the periphery or middle of either lung and are sharply defined at the start and become ill-defined as the infection spreads to the periphery.

There is no agreement as to the treatment except that the majority feel medical treatment, with rest, diet and postural treatment is indicated for 2 to 3 months and if no improvement occurs, some form of surgery is indicated. Of course if fusospirochetal organisms are found, arsphenamine should be given early.

Surgical procedures include mainly bronchoscopy, pneumothorax, pneumolysis and two-stage plastic operation.

One might reasonably hope for a reduction in the number of lung abscesses, especially those secondary to tonsillectomies, if proper oral hygiene were instituted prior to the removal of the tonsils and provided the operator is skillful and examines his patients not only a few weeks previously, but on the day of the operation.

PLASMA CELL MYELOMAS CAUSING CORD COMPRESSION: REPORT OF FIVE CASES*

By ROLAND M. KLEMME, M.D.,
St. Louis, Mo.

The literature contains numerous reports of various types of extramedullary tumors causing compression of the cord. In all of these reports there are but few instances of myelomas producing such symptoms. Elsberg, in his text book on tumors of the spinal cord, states he has seen 3 patients with myelomas, and in Frazier's 330 cases he found but 3 myelomas. For this reason it seems worth while to report 5 cases of this kind observed in the past three years, and to draw attention to some important features.

First, in all but one of these cases the preoperative diagnosis was spinal cord tumor. In none was a myeloma suspected, though a bony tumor was recognized in one roentgen plate, but the lesion was thought to be actinomycosis, and though myelomas in other bones are readily recognized by the roentgen ray, in these cases there was no evidence of any bony change in the vertebrae. Second, in all these cases there was no evidence at the time of operation of any other focus in any of the other bones in the body. Third, when completely removed at operation and followed up by intensive deep roentgen therapy, the results are very satisfactory.

*Read in Section on Neurology and Psychiatry, Southern Medical Association, Twenty-Sixth Annual Meeting, Birmingham, Alabama, November 15-18, 1932.

*From the Department of Neurological Surgery, Washington University School of Medicine.

In the past three years, 5 cases of plasma cell myeloma presenting signs of cord involvement of a paraplegic kind have come under my care. The tumor mass in all of these cases was verified at operation. Four cases were in men and one in a woman.

REPORT OF CASES

Case 1.—J. B., age 46 years, was admitted to Missouri Baptist Hospital on January 21, 1929, complaining of numbness in the pit of the stomach, tingling and marked weakness in both legs, more on the left; inability to walk, constipation and retention of urine. The family history and past history were irrelevant. The onset of his present complaint began four weeks prior to the admission, at which time the patient noticed a tingling in the left leg; this gradually spread to the right leg. One week later the patient noticed marked weakness in the left leg, and several days later the weakness extended to the right leg. Weakness in the legs became progressively worse until the patient was unable to walk.

Neurological examination showed a loss of sensation below the fifth dorsal spine segment, with an associated spastic paraplegia. There was a bilateral ankle and patellar clonus, and a bilateral Babinski, Oppenheim, and Chaddock. A Queckenstedt test showed a complete block. The spinal fluid was clear and colorless. Position sense and vibratory sense were intact. The roentgen plates of the spine showed no bony change in the vertebrae of the dorsal spine. The urine examination showed no Bence-Jones bodies present.

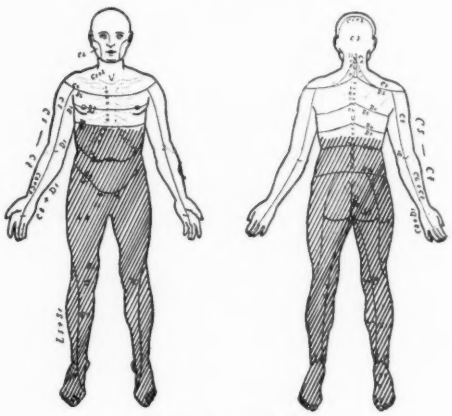

Fig. 1, Case 1, J. B.
Shaded area signifies loss of cutaneous sensations.

No.	Sex	Age	Duration of Illness	Onset	Clinical Diagnosis	Operation	Response to X-Ray	Metastasis
1. J. B.	Male	46	4 weeks	Pain	Cord Tumor	Laminectomy	Excellent	None
2. W. D. D.	Male	56	10 months	Pain	Cord Tumor	Laminectomy	Good	Rt. humerus in 1 year Lt. humerus in 2 years
3. F. H. N.	Male	67	14 months	Pain	Cord Tumor	Laminectomy	Died	None
4. C. W.	Female	33	6 months	Pain	Cord Tumor	Laminectomy	Good	None
5. O. J. N.	Male	28	7 months	Pain	Actinomycosis	Laminectomy	Good	None

Operation.—On February 5, 1929, a laminectomy was done with a removal of a large extradural, very vascular, reddish tumor in the region of the fifth dorsal spine segment that had completely compressed the cord. Postoperative course was uneventful, and on February 7, 1929, the patient began to move his legs and toes; five days later he was up in a chair, and on February 18, 1929, he was walking with a cane.

He received deep roentgen therapy on February 19 and 20, 1929, and another series of treatments was given on April 8, 1929. In June, 1929, the patient was able to discard his cane, and has been working every day since. This patient received more roentgen therapy on April 27 and 28, 1932, and again on July 13, 1932.

At no time has the patient shown any signs of metastasis to any other part of the body.

Since June 18, 1929, the patient has been very active and has been able to carry out his occupation perfectly normally to date.

Case 2.—W. D. D., age 56 years, was admitted to Barnes Hospital on August 15, 1931, complaining of pain in the upper portion of the back, which started ten months prior to the admission. In April, 1931, the patient noticed a numbness of his feet. This gradually and progressively ascended to above the umbilical region. Four months prior to the admission he noticed increased difficulty in urinating. Since the onset of the numbness, the patient had difficulty in walking. The general weakening of the legs increased and the patient had to use crutches, and one month prior to the admission was unable to walk at all.

Neurological examination showed a complete loss of sensation to pain, touch and temperature extending up to the level of the fifth dorsal spine segment. There was a definite and marked sweating above the lesion. Associated with these findings there was a spastic paraplegia, bilateral ankle and patellar clonus, bilateral Babinski and Oppenheim. A Queckenstedt test done at that time showed a complete block; the fluid was xanthochromatic. The protein content of the fluid was 250 milligrams per cent. Associated with these findings this patient had a polycythemia; a red cell count ranging from 7,200,000 to 7,800,000. The urine examination showed no Bence-Jones bodies. The roentgen pictures of the spine showed no change in the bone other than a slight curvature.

Operation.—On August 18, 1931, a laminectomy was done over the third, fourth and fifth spinous processes, and a tumor about the size of a lemon was removed between the third and fifth dorsal vertebrae completely compressing the cord at the fourth and fifth dorsal spine segments. The tumor was very vascular, but showed no evidence of invasion of the meninges. On August 26, 1931, the patient moved the toes of his right foot, and the motion in his legs rapidly recovered. The postoperative course was uneventful, and on September 10, 1931, the patient was able to walk with the aid of a cane.

This patient received deep roentgen therapy on September 1 and 2, 1931, and again on October 23, 1931. At the time of his last treatment the patient complained of pain in the left humerus. An x-ray picture of the humerus showed a myeloma of the bone at the lower third. The patient was given deep roentgen therapy to the arm, as well as to his spine. He returned to the hospital on October 20, 1932, with a pathological fracture of the right humerus. The roentgen examination of the right humerus showed a myeloma of the lower third. The patient was given deep roentgen therapy to the right humerus and also to his dorsal spine. The roentgen picture of the left humerus at this time showed no evidence of myeloma. This patient did not return for deep roentgen therapy as advised, which undoubtedly influenced his course.

Case 3.—F. H. N., age 67, was admitted to Missouri Baptist Hospital on September 14, 1931, complaining of inability to walk. His family history and past history were irrelevant. The present illness began about one year prior to his admission, when he noticed a beginning numbness in both legs, associated with inability to use his legs. The motion in his legs gradually became worse and one month prior to admission to the hospital he was unable to walk.

Neurological examination showed a definite sensory level with loss of all sensations to pain, touch, and temperature below the fourth dorsal skin segment. There was definite tenderness over the first dorsal

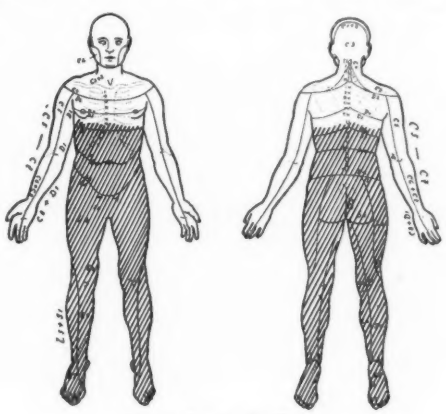

Fig. 2, Case 2, W. D. D.
Shaded area signifies loss of cutaneous sensations.

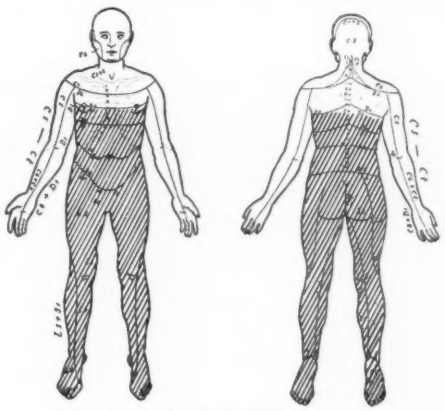

Fig. 3, Case 3, F. H. N.
Shaded area signifies loss of cutaneous sensations.

spine. In addition to this, the patient had a spastic paraplegia with a bilateral ankle and patellar clonus, and a bilateral Babinski. A Queckenstedt test showed a complete block. The fluid was clear and colorless. The protein content was 169 milligrams per cent. The roentgen examination of the spine showed no change in the vertebrae.

Operation.—The patient was operated upon September 21, 1931. A laminectomy was done and a tumor was removed from the spine at the level of the seventh cervical vertebra. The tumor was very vascular and compressed the cord.

On September 22, 1931, the patient showed evidence of an acute heart failure with an associated edema of the lungs, and he expired on September 23, 1931.

Case 4.—C. W., age 33, was admitted to Jewish Hospital in April, 1932. On May 1, 1932, she was transferred to my service. This patient complained of numbness of the lower extremities, which started in April, 1932. Three days later she was unable to walk because of weakness in her legs and spasticity. On May 5, this patient noticed for the first time retention of urine and marked constipation. At no time did she complain of any pain.

Neurological examination showed tenderness over the seventh dorsal spinous process and a sensory change extending from the junction of the eighth and ninth dorsal skin segments downward. There was an associated spastic paraplegia with bilateral ankle and patellar clonus, and a bilateral Babinski and Oppenheim. A lumbar puncture was done and the fluid was clear and colorless. A Queckenstedt test showed evidence of the blocking of the spinal canal. The roentgen plates of the spine showed no evidence of bone change. The urine examination was entirely negative.

Operation.—On May 25, 1932, a laminectomy was done and the fifth, sixth, seventh and eighth dorsal spinous processes were removed. A tumor 4 cm. long and 2.5 cm. in diameter was removed at the level of the seventh and eighth dorsal spine segments. The tumor was very vascular and almost completely compressed the cord. On June 8, 1932, this patient was given deep roentgen therapy, and again in August, 1932. At the end of July, 1932, the patient was up in a chair and was able to get around with assistance. The patient's course since that time has been one of steady improvement.

Case 5.—O. J. N., age 28, was admitted to Barnes Hospital on June 4, 1932, complaining of pain in his neck. This pain was accentuated by the movements of his head and grew progressively worse. At times the pain radiated out to his shoulders. Four to five months prior to the admission the patient was confined to his bed because he could not lift his head. From that time on, the pain in his neck has been continuous.

Neurological examination showed marked tenderness from the fifth cervical to the second dorsal spinous processes. There was no demonstrable sensory change, nor was there any paralysis present. However, his knee jerks and ankle jerks were hyperactive, although no pathological toe signs could be elicited. A spinal puncture and Queckenstedt test were done. The spinal fluid was clear and colorless. There was no evidence of a block in the canal. The protein content of the spinal fluid was 16 mg. per cent. The urine analysis showed no evidence of the presence of Bence-Jones bodies. The roentgen picture of the cervical and dorsal spine showed a destructive process of the sixth and seventh cervical vertebrae and an involvement of the transverse processes of the first, second, and third dorsal vertebrae. The destruction involved the attachment of the ribs at these points. The roentgen diagnosis, as well as the clinical diagnosis, was actinomycosis of the vertebrae.

Operation.—On June 13, 1932, a laminectomy was done, which extended from the fifth cervical to the second dorsal spinous processes. A fairly large mass was removed and the last cervical as well as the first dorsal spinous process was entirely eroded. The postoperative course was uneventful.

The patient was given deep roentgen therapy on July 14 and 15, August 28 and 29, and October 26 and 27, 1932, and was discharged from the hospital after a Thomas collar had been constructed to support his head.

Pathology.—The tumor material in each of the above cases was fairly firm, had a deep red color, and showed

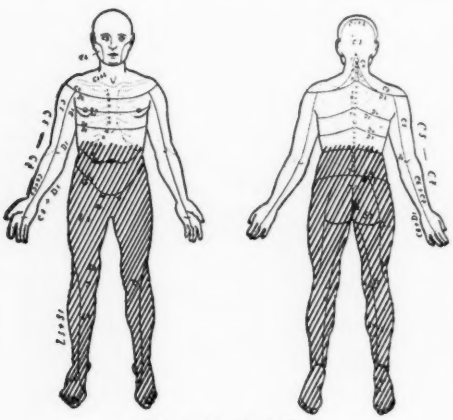

Fig. 4, Case 4, C. W.
Shaded area signifies loss of cutaneous sensations.

Fig. 5
Plate of cervical spine showing destruction of bone in lower cervical and upper dorsal vertebrae.

Fig. 6
Metastatic myeloma to right humerus one year after operation (*Case 2*).

Fig. 7
Metastatic myeloma to left humerus two years after operation with pathological fracture (*Case 2*).

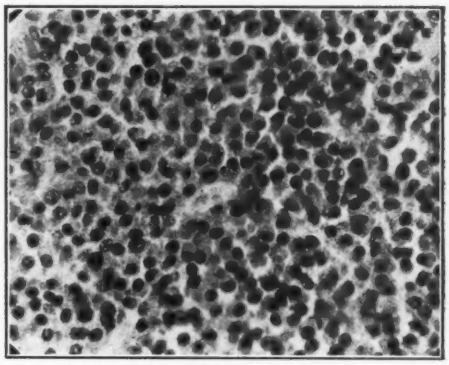

Fig. 8
Section of typical plasma cell myeloma. Hematoxylin and eosin stain.

marked vascularity. Microscopic examination of the tumor material from each specimen showed loosely packed typical plasma cel's, round, oval, and polygonal with non-granular cytoplasm. Mytotic figures were present in each section, but were not abundant. In none of these cases was there a direct invasion of the spinal cord or meninges. The symptoms were due to compression of the cord by the growth in the vertebrae.

CONCLUSION

In the presence of a focal spinal lesion, even though it show no characteristic x-ray findings, the possibility of an isolated myelomatous lesion has to be kept in mind. A positive diagnosis cannot, of course, be made until the tissue has been removed at operation. The absence of other bone lesions does not preclude the possibility of a myeloma.

If these cases are treated after operation intensively at regular intervals with deep roentgen therapy, the prognosis in most cases should be good.

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DISCUSSION (*Abstract*)

Dr. R. G. Spurling, Louisville, Ky.—Dr. Klemme's report is unique in at least two respects: first, he has had many cases of plasma cell myeloma in a brief space of time; and second, the results have been encouraging.

This is a re'atively rare tumor. On looking over our series of spinal cord tumors for the past five years, I find that we have had only two instances of proven plasma cell myeloma. Both of these patients were advanced cases. One was operated upon inadvertently and died several days later. The other was in a hopeless condition and was not subjected to operation. Both patients were autopsied and extensive destruction of the vertebrae was found. One showed Bence-Jones protein; the other did not.

On looking over the available literature on the subject, I find that irradiation has been found in the past to be merely a palliative procedure with this type of tumor. It is indeed encouraging to know that life may be extended over a long period by the treatment which Dr. Klemme outlined.

Dr. Klemme (closing).—Isolated myelogenous lesions are not uncommon and must be thought of in the presence of a focal spinal lesion. Deep roentgen therapy given intensively at regular interva's has controlled the condition in the group reported above.

ASTHMATIC BRONCHITIS: TREATMENT WITH IODIZED OIL (LIPIODOL)*

By JAMES F. ALISON, M.D., F.A.C.P.,
Selma, Ala.

All types of asthma have come to be regarded as an expression of an allergic reaction. The exciting allergin may be food, bacteria, animal or plant emanations. Cooke[1] estimates that approximately one-third is bacterial or infectious in origin. He also states that in 10 per cent of this group the primary focus is in the bronchi. However, it is reasonable to assume that bronchial involvement is the ultimate and inevitable result of any chronic upper respiratory infection. The primary focus may be in the mouth, paranasal sinuses or pharynx. Removal of the foci has been disappointing as to curative value.

Ochsner,[2] in his excellent work with iodized oil in the diagnosis and treatment of bronchiectasis, has uncovered two important facts which form the basis for this study. First, he showed that in chronic bronchitis there is almost invariably a change in the terminal bronchi. This change varies from a slight dilatation to bronchiectasis. Second, he demonstrated that, by actual count, iodized oil caused a decrease in the number of bacteria in the lesion thus treated.

Assuming that asthmatic bronchitis is due to sensitization to the infecting bacterial protein, it is rational to direct treatment toward the eradication of the exciting agent. In accord with this line of reasoning, I began treating a group of patients who suffered this type of asthma by introduction of lipiodol into the tracheobronchial tree. The initial response in all patients was striking. Practically all gained symptomatic relief within twenty-four to forty-eight hours. The period of freedom from symptoms varied with the individual. I found that the patients who had the most marked change in the bronchi, short of frank bronchiectasis, seemed to benefit most from the treatment. Those patients with bronchiectasis gained relief, but required rather frequent treatments to remain symptom-free. I was unable to duplicate the results obtained by Fink,[3] who found that one treatment was sufficient in some patients apparently to cure the asthmatic symptoms. Ochsner[4] has observed the beneficial effects in a number of patients and thinks that it is probably the most efficacious treatment of asthmatic bronchitis, especially if there is an accompanying bronchiectasis. The treatment consisted of introduction of lipiodol by the passive method described by Ochsner. No difficulties or untoward effects were noted. The interval between the treatments and their number were governed by the therapeutic response, some patients receiving only one while others required treatments every three to ten weeks to remain symptom-free. It was usually possible to lengthen the interval after the patient had taken two or three.

The patients ranged from eight to sixty-five years in age. The asthmatic symptoms had been present from one to fifteen years. Four were bed-ridden and two were dangerously ill when first treated. All were benefited and some have remained clinically well for a year or more. No patient complained of the treatment and they submitted cheerfully to repeated injections.

CASE REPORTS

Case 1.—Mr. D., a farmer of 63 years, had suffered asthmatic attacks for the past ten years. For the previous six months the attacks had been more frequent and severe. For several weeks prior to consultation he had been incapacitated and was compelled to sleep sitting up. Examination showed severe oral sepsis, chronic bronchitis and hypertensive heart disease. Lipiodol was introduced and the chest plate showed definite dilatation of the lower bronchi. He reported two weeks later much improved, the attacks much less severe and frequent. He was treated four times at intervals of 2, 3, 6 and 8 weeks, respectively. Four months after the last treatment he reports that he is symptom-free, able to perform his farm duties and has gained 15 pounds in weight.

Case 2.—A. B., a schoolboy of 8½ years, has suffered asthmatic attacks for the previous four years. The paranasal sinuses were normal, the tonsils had been removed and the skin tests proved negative. The attacks had become so severe as to require morphia for relief. For two weeks before the first treatment he was unable to attend school and required one to three hypodermics a day. He improved immediately after the first injection of oil and needed no more epinephrine or morphia. He requires treatments every two to three months to remain symptom-free.

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*From the Marcus Skinner Clinic.

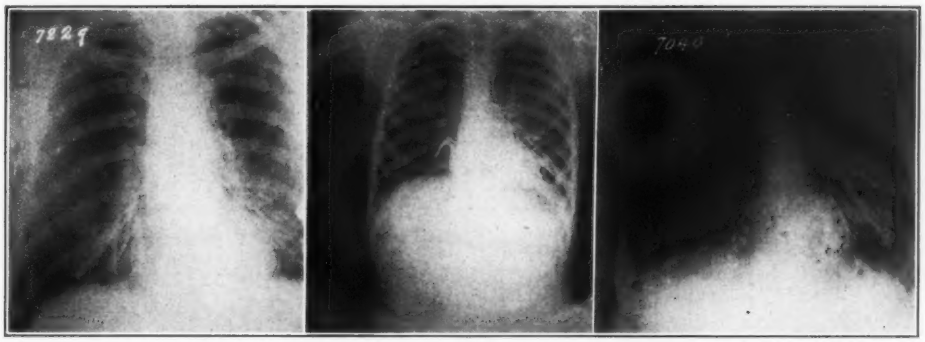

Fig. 1, Case 1

Extensive bronchial changes. Cylindrical and saccular dilatation.

Fig. 2, Case 2

Slight cylindrical dilatation of bronchi. Although the departure from normal was slight, excellent results were obtained with this patient.

Fig. 3, Case 3

Marked cylindrical dilatation. Some sacculation. Excellent results.

Case 3.—Mrs. B., a housewife of 59 years had suffered severe attacks of asthma for the previous three years. For the previous three months she had suffered constantly, being confined to her bed most of the time. During her illness she had lost 40 pounds in weight. Examination showed chronic bronchitis, but no other findings of note. She had a productive cough and raised considerable sputa. Lipiodol was injected and the plate showed definite widening and slight sacculation of the lower bronchi.

During the preceding nine months she has been given 5 injections with almost complete relief. She has gained 32 pounds in weight and is able to perform her usual household duties.

Case 4.—Mr. S., a university student, has suffered with asthma for five years. No etiological factor has been discovered although he has been thoroughly examined. Lipiodol was introduced as a therapeutic measure and the chest plate showed some saccular dilatation of the terminal bronchi, a condition which was unsuspected. He obtained considerable relief but continued to have mild attacks. One month later a second injection was given with subsequent relief for six weeks, when the attacks began anew. He is being treated at intervals of 6 to 8 weeks which serves to keep him symptom-free and allows him to participate in athletic activities, which he has been unable to do until recently. He feels that he will ultimately be cured.

Case 5.—P. G., a negro man of 49 years, had periodic attacks of asthma for the past five years. During recent months he suffered a cardiac breakdown, auricular fibrillation with congestive heart failure. Since this occurrence his asthmatic attacks increased in severity and frequency. For two weeks before he was referred, he required hypodermics of morphia or epinephrine or both at intervals of 3 to 6 hours. It was questionable whether the distress was asthmatic or cardiac but, on account of the patient's extreme discomfort, I felt that any measures were justified. He was injected while on the ambulance cot and a chest picture was made. The heart and great vessels were immensely enlarged and the lower bronchi showed sacculation and cylindrical dilatation. Within twenty-four hours he was able to breathe much better and although his cardiac condition progressed, he required no more epinephrine and was free of asthma for three months when he reached a cardiac terminus. This was especially interesting, as he had never gone more than a week in the past four years without epinephrine.

Case 6.—Mr. C., a farmer, had enjoyed excellent health until a year before our examination when he began having asthmatic attacks which followed a severe attack of influenza. These attacks gradually increased in frequency and severity until the symptoms were present almost constantly. Plates after lipiodol showed cylindrical dilatation of the lower bronchi. Twenty-four hours after the first treatment he was symptom-free. The attacks returned in three weeks when he received a second injection. He is able to go from 6 to 8 weeks between treatments in comfort.

Case 7.—Mr. D., a clerk of thirty years, had suffered with asthmatic attacks for the previous year. He had changed jobs because he thought the dust of the office might be responsible. I saw him eighteen months ago and gave him an injection of lipiodol. He has suffered no asthma since and gained 31 pounds in weight. His plate showed considerable evidence of bronchial dilatation. This is the only patient who required only one treatment.

Case 8.—Mrs. N., a woman of 45 years, who has a pronounced bronchiectasis with profuse expectoration, developed asthmatic attacks within the last year. These attacks have become severe and distressing. The picture with lipiodol instilled showed several bronchiectatic cavities. Her symptoms, especially the asthma, were markedly benefited by repeated injections of lipiodol. She requires treatments at intervals of 1 to 3 months.

Case 9.—Mrs. W., a housewife of 63 years, suffered with asthmatic attacks for three years. The attacks gradually increased in severity. Injection of lipiodol showed marked dilatation of the lower bronchi. Three injections were given at intervals of 3 to 8 weeks. Six months after the last treatment she is still symptom-free.

Case 10.—Mrs. N., a housewife of 39 years, had suffered with periodic severe attacks of asthma for some three years. For four months before examination she had been bedridden and had lost considerable weight. The only physical findings of importance were malnutrition and chronic bronchitis. After the first filling she improved markedly, gained twelve pounds in weight and was up and about. She then had a severe attack of influenza and the asthma returned. The second and third injections of lipiodol gave very little relief. She is better than before the treatment but not relieved and has to seek other measures for her distress.

DISCUSSION

The bronchograms of these patients all show some deviation from the normal. This varies from slight dilatation to extensive processes as illustrated in *Case 8*. The most spectacular results were those obtained in the negro who had

Fig. 4, Case 8

Bronchiectasis. The lower bronchi are markedly deformed.

Fig. 5, Case 9

Saccular and cylindrical dilatation of the lower bronchi.

Fig. 6, Case 10

Considerable bronchial changes. Results in this patient were only fair. The treatment was abandoned as not worth while.

Table 1

SUMMARY OF PATIENTS TREATED WITH LIPIODOL. NOTE THAT ALL PATIENTS SHOWED ABNORMAL BRONCHOGRAMS

Patient	Age	Duration of Asthma	Roentgenologic Findings	Number of Treatments	Results	Remarks
W. D. 1	63	10 years	Dilatation of lower bronchi	3, at intervals of 1 to 2 mos.	Excellent	No attacks since second treatment. Gained 10 pounds. Able to do farm work for first time in 3 years
A. B. 2	10	3 years	Cylindrical dilatation of bronchi	2, 6 week interval	Excellent	Mild attack after first treatment. No attacks since second treatment. Gained 8 pounds. Back in school
Mrs. B. 3	59	4 years	Definite bronchiectasis	4, intervals of 2 to 4 mos.	Excellent	Was bed-ridden; improved quickly; gained 28 pounds in 2 months; able to work
D. S. 4	19	5 years	Slight dilatation of bronchi	3 in 4 mos.	Excellent	Has occasional mild attack which requires no medication
P. G. 5	52	2 years	Dilatation of bronchi. Enormous heart	One	Excellent	The asthmatic attacks ceased 24 hours after treatment. Did not recur. Heart fatal 3 mos. later, but had no more asthma
I. C. 6	31	1 year	Cylindrical dilatation of bronchi	2, 6 week intervals	Excellent	Clinical cure. No asthma after the second treatment
S. D. 7	32	18 mos.	Sacculation of terminal bronchi	One	Excellent	No attacks since first treatment 8 months ago
Mrs. N. 8	45	15 years	Advanced bronchiectasis	5, intervals of 1 to 3 mos.	Good	Patient requires treatments every 1 to 3 months to remain symptom-free. Large bronchiectatic cavities present
Mrs. W. 9	54	4 years	Saccular and cylindrical dilatation of bronchi	Three	Excellent	No attacks for ten months after last treatment. Before, the attacks occurred almost daily
Mrs. M. 10	39	3 years	Marked dilatation of bronchi	3, intervals of 1 to 3 mos.	Fair	Patient improved after first treatment, then had severe influenza and asthma returned. Improved, but not relieved

severe heart disease. He was more comfortable twenty-four hours after the injection than he had been in weeks. This comparative comfort continued until his exitus three months later. I cannot explain the lack of relief experienced by *Case 10*. The bronchogram showed considerable deviation from the normal and she had profuse expectoration. It is probable that her attacks were extrinsic in origin. I do not think that lipiodol instillations are of value except in selected cases, namely, those patients who have a bronchial lesion and who are sensitive to the infecting protein.

CONCLUSIONS

(1) In selected cases lipiodol instillations are of unquestioned value in treating asthmatic bronchitis.

(2) Repeated instillations, the intervals being governed by the therapeutic response, are necessary in the great majority of patients.

(3) As a rule, patients with the most bronchial disease, short of extensive bronchiectasis, obtain the greatest symptomatic relief, the intervals between treatments being longer.

(4) I believe that the beneficial effects of lipiodol installations lie in their ability to exert a bactericidal action upon the infecting microorganism.

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STUDIES ON THE METABOLISM AND THE RESULTS OF TREATMENT IN VARIOUS FORMS OF ARTHRITIS*

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Before presenting to you the metabolic studies made on a series of 65 patients suffering from various forms of chronic arthritis, it will be necessary to explain in some detail the basis for classification.

The pioneers in this field were Nichols and Richardson.[1] In 1909, they reported in the *Journal of Medical Research* a series of pathological studies of fundamental importance on chronic arthritis. After eight years of continued work they felt that it was possible to classify all cases under two heads: proliferative and degenerative. They did not feel that it was possible to make any definite statements as to etiology except that:

"Traumatism, acute suppurative infections, gonorrhea, syphilis, probably also a variety of other things may induce primary proliferation of the synovial membrane," and might be the basis for the proliferative type; "while old age, traumatism, dislocations, the presence of tumors of bone, gout, disease of the central nervous system and other causes we have seen lead to primary degeneration of the cartilage," and these might be the basis for the degenerative changes.

In order to establish a basis for following the changes, a more or less diagnostic representation of a joint has been reprinted from their article in *Fig. 1*.

Their summation of the fundamental changes is so well done that I have taken the liberty of quoting several paragraphs, as follows:

"*Proliferative Arthritis.*—In this type of joint lesion the primary change occurs as a proliferation of the synovial membrane and of the perichondrium of the articular cavity, combined in many cases with a synchronous proliferation of the connective tissue and endosteum of the epiphyseal marrow directly below the joint cartilage. The most marked change usually is in the synovial membrane, while the extent to which the perichondrium proliferates is variable, being in many joints slight and almost negligible, while in other cases it is very marked. As a result of the proliferative changes of the synovial membrane and of the perichondrium, two sorts of changes take place within the joint. The proliferation of the synovial membrane produces a layer of granulation tissue, which sooner or later may extend over the joint cartilage as a thin pannus-like layer. Where this synovial pannus comes in contact with cartilage, it usually produces a destruction and absorption of the cartilage. Whenever a proliferation of the perichondrium takes place, however, it leads to the formation of a layer of specialized connective tissue, which readily transforms itself into cartilage or even into bone. Consequently, two processes may go on in these joints simultaneously, namely, a destruction of the joint cartilage by synovial pannus, and a new formation of cartilage or bone arising from perichondrial proliferation. The relative amount of destruction or of new formation varies in different joints and with different causes. The important fact is that the two processes may go on simultaneously. In addition to these two processes which take place within the joint cavity analogous changes may take place within the marrow of the epiphysis of the bone itself. There may arise a proliferation of the connective tissue of the marrow spaces of the epiphysis just below the zone of provisional calcification. This proliferated connective tissue of the marrow also is accompanied with the formation of numerous blood vessels. This vascular granulation tissue may extend through the zone of provisional calcification upwards to the joint and cause a

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destruction and disappearance of the overlying cartilage in two ways: from the joint surface by the action of synovial pannus and from the perichondrium by the action of the granulation tissue of the marrow (*Fig. 2*).

"Usually besides this proliferation of the connective tissue of the marrow there also is present a proliferation of the endosteum of the epiphysis which may result in the formation of new bone cartilage along the epiphyseal margin of the articular surface." (True ankylosis may take place in this type of case.)

"*Degenerative Arthritis.*—General process. In this class of joint lesion the earliest and primary change in the joints is a degeneration of the hyaline cartilage of the articular surfaces (*Fig. 3*). As a result of this cartilaginous degeneration, the cartilage becomes softened and eroded, and the underlying bone may become exposed so that instead of two cartilaginous surfaces being in contact the cartilage of one of the bones of the joint may be in contact with exposed bone, or two bone surfaces may articulate. Since the degeneration of cartilage usually is not uniform over the whole extent of any given joint, the areas of exposed bone at first generally are circumscribed. Wherever erosion of cartilage or of bone occurs there practically always comes a compensatory growth of cartilage or of bone of the opposite articular facet so that the articulating surfaces of the two bones of the joint always are in contact, although as a result of the erosion of one bone and a corresponding overgrowth of cartilage due to proliferation of the perichondrium of the opposite articular surface, the line of the articulation becomes very irregular (*Fig. 4*). In most cases the fibrillation of the cartilage at first affects only a portion of the articular facet of one bone, but ultimately may extend over the entire joint surface of both of the bones which enter into the articulation, so that ultimately the entire articular cartilage may disappear from both bones. So that, finally,

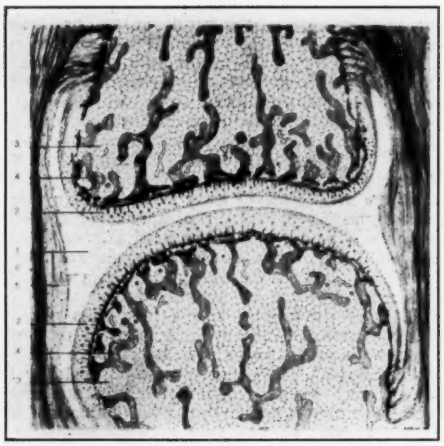

Fig. 1

(From the *Journal of Medical Research*, Vol. 21, Plate IV, Fig. 1.) Drawing, semidiagrammatic, to illustrate the histology of a normal joint. Drawing represents a vertical section through the phalangeal joint together with the articular ends of the adjacent phalanges. (1) is the joint cavity; (2, 2) are the articular cartilage; (3, 3) show the fat marrow; (4, 4) are trabeculae; (5) is synovial membrane; (6) is fibrous capsule.

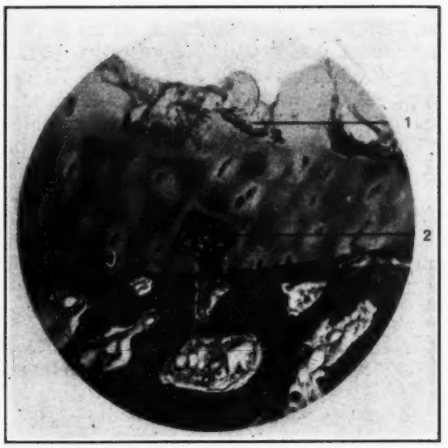

Fig. 2

(From the *Journal of Medical Research*, Ibid, Plate X, Fig. 19.) Proliferative arthritis; fairly early stage. Gonococci found in the joints. From a woman thirty-three years of age who was admitted to the hospital with acute pelvic inflammation which was followed two weeks later by swelling of the elbow, wrist, and left knee successively, with pain and fixed joints. Finally the knee was straightened under ether, but the pain continued and the knee joint was opened and drained, and finally was excised. Photomicrograph of a section of the articular cartilage of the lower end of the femur, at right angles to the joint cartilage. The articular surface is covered by a thin layer of synovial pannus (1), beneath which the surface is eroded. The granulation tissue of the epiphysis is extending upward into the cartilage.

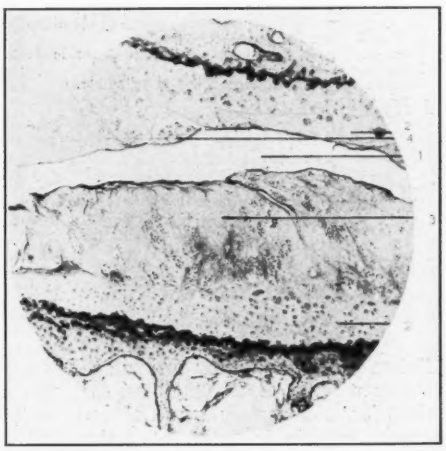

Fig. 3

(From the *Journal of Medical Research*, Ibid, Plate XVIII, Fig. 49.) Degenerative arthritis, early stage. Photomicrograph of section of phalangeal joint and adjacent phalanges, high power. Shows joint cavity (1); joint cartilages (2, 2). There is vertical fibrillation of the matrix (3) of the cartilage, with absence of staining reaction of the cartilage cells, over the greater portion of the upper two-thirds of the cartilage. The hypertrophic zone of cartilage cells is normal. One small island of cartilage (4) is broken off and lies free in the joint.

instead of two cartilaginous surfaces being in contact, two bony surfaces are in contact, but since this change is brought about gradually, and since in the early stages an erosion of one bone is met by a corresponding overgrowth of the opposite facet, the two bony surfaces which result are very irregular in outline and in many cases present an extremely notched appearance. As a resu't, the exposed bone becomes extremely dense and acquires a structure as solid as that of normal cortical bone, or may be even denser. This exposed dense bone under the friction of continued joint motion acquires a high degree of polish, and has an appearance closely resembling that of ivory, hence the term "eburnation of bone." . . . . As a result of the changes in the shape of the joint surface the plane of the articulation may be changed and the amount of joint motion may be very much diminished in certain cases; in fact, the irregularity of the joint surface may become so great that the two bones may become interlocked and no motion may be possible, although there is no true anchylosis of adjacent joint surfaces."

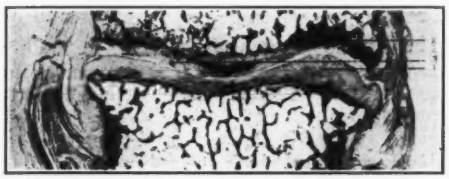

Fig. 4

(From the *Journal of Medical Research*, Ibid, Plate XVII, Fig. 43.) Degenerative arthritis; moderate degree; cause unknown. Photomicrograph of section through a phalangeal joint and adjacent phalanges shows that the line of the joint cavity is very irregular. Areas of hyperplasia of the cartilage (1), with, in other places, erosion of the cartilage down to eburnated bone of the opposing phalanx (2). In other cases the cartilage shows fibrillation (3). There was moderate thickening of the capsule.

This work has been corroborated by Strangeways[2] and by Fisher.[3]

From an increasing number of x-ray investigations two main types of chronic arthritic joints have been established. The first, the atrophic type, is one in which the joint shows fusiform swelling with at times distention of the joint cavity due to fluid. The change within the joint usually causes a hazy or flakey appearance. It may be associated with areas of punctate destruction (*Fig. 5*). The epiphysis of the bases are usually more permeable to the rays due to absorption of lime salts. Many workers believe this to be a disuse atrophy. It is quite suggestive that these changes are due to the proliferative type of arthritis and may be explained on an infectious basis.

Fig. 5, Case 19
Infectious arthritis of the little finger.

Fig. 6, Case 38
Hypertrophic arthritis of the knee.

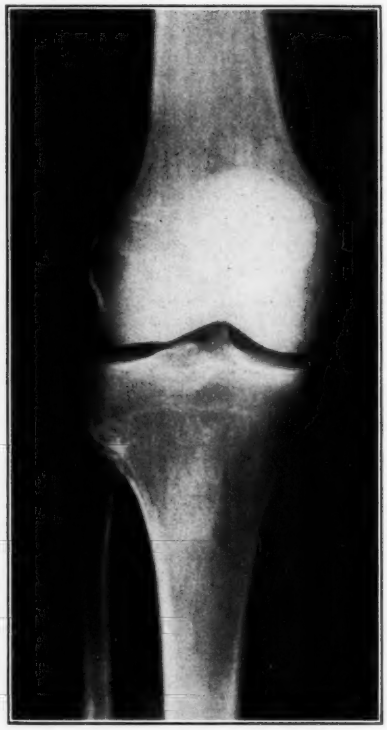

Fig. 7, Case 35.
Spicules at the insertion of the tendons.

The second type of x-ray change is found in the hypertrophic group. Here bone deposits are found at the articular line of cartilage and epiphysis, but within the joint cavity (*Fig. 6*). Spicules of bone are also found at the insertion of tendons where they assume a stalactite or stalagmite appearance (*Fig. 7*). Cyst-like areas of decreased density may be seen honeycombing the new bone deposits (*Fig. 8*). Frequently along the epiphyseal line it is possible to see a bony layer of increased density similar to the area of eburnation, described by Nichols and Richardson.[1] The hypertrophic x-ray group corresponds in many ways to the degenerative group of these authors.

At this time it is impossible to state that the two x-ray groups, atrophic and hypertrophic, are always representative of the proliferative and degenerative types. It is quite possible that early stages of the degenerative type might give x-ray findings completely in accord with early damages in the so-called atrophic x-ray type. Furthermore, certain of the later stages of the proliferative type may give x-ray evidence indistinguishable from the hypertrophic variety. A consideration of these problems demonstrates the difficulty of classifying patients by any one finding.

It is generally accepted that the great bulk of cases fall clinically (from an etiological viewpoint) into two main types: the infectious or rheumatoid arthritis and the metabolic or osteoarthrosis.

The infectious type usually occurs in youth or young adults. In childhood it has long been known as Still's disease. Its onset is frequently acute, although it may be insidious. It rapidly becomes poly-articular and is characterized by fusiform swellings which are red, warm and tender. In the early stages there is no x-ray evidence of joint involvement. It is quite possible, however, that at this time the pannus-like inflammatory mass originating from the synovial membranes is destroying areas of the cartilage and forming fibrous adhesions between the two bones.

Infection, although it may not be the sole etiological factor in the production of the proliferative type of arthritis, is certainly an outstanding one. As mentioned above, the changes produce the atrophic x-ray findings, and as the disease progresses and as more bone is laid down by

Fig. 8, Case 44
Hypertrophic arthritis with cyst-like areas of the hip joint; a mono-arthritic.

conversion of cartilage, it is possible to make films which demonstrate very similar findings to those of the rare types of hypertrophic arthritis.

The metabolic type of the disease usually occurs in later life, although this is by no means an unbroken rule. The onset is more apt to be insidious than in the infectious type, although, here again, exceptions are all too common. The disease, in the early stages, is more apt to be mon-articular and accompanied by less rugor, calor and dolor than in the other group. X-ray films taken at this stage may show nothing or may show any grade of bony deposit within the joint cavity. It is amazing to compare the short duration of symptoms with the length of time which must have elapsed during the formation of bony deposits. At other times great distress with marked limitation of joint function has been found where very small or no x-ray change could be demonstrated. In these instances the etiological factor must, in some manner, be irritating the synovial membrane.

Many of the above remarks are premature because they have been based partly upon certain studies on the metabolism of chronic arthritics and interpreted from their clinical and x-ray findings. The results of treatment have, in no small degree, also had their influence.

The findings to be reported were made on a group of 65 patients; the larger number of these were referred by Dr. George E. Bennett, of Baltimore, because he believed that some metabolic disturbance was an etiological factor. In most instances he committed himself to this diagnosis, but occasionally the case was studied because he felt unable to place it in either group. The tabulation of results both from the point of view of metabolic study and from therapy shows a more than creditable judgment on his part.

The patients were all given a thorough diagnostic study, although needless to say the great majority of them had had similar examinations before. Most of them had been carefully searched and surgically treated for alleged foci of infection. Many had been baked, or strapped or splinted. Various sera, vaccines and toxins had been given without avail.

With but few exceptions, sugar tolerance tests, purine metabolic tests, basal metabolic determinations, gastric analyses, fluoroscopy of colon for position, outline and function and the routine blood, urine and stool studies were made on all cases.

For many years since the work of Burian and Schur[4] it has been believed that approximately 50 per cent of the ingested purine nitrogen is excreted as uric acid nitrogen.

The more recent work of Folin, Berglund and Derick[5] has brought new light upon the subject. A normal individual was placed on a purine low diet until the endogenous urinary excretion of purine became stationary. They then found, after intravenous injections of uric acid (amounts varying from one to two grams), a marked increase in blood uric acid which held for periods of two or three days. There was also an elevation in the urinary output of uric acid for several days before it returned to the former endogenous level. From their tables I have plotted their results in *Fig. 9*. The points of interest are:

(a) The endogenous excretion varied between 400 and 600 mg. per 24 hours.

(b) The amount of injected uric acid recovered in the urine varied between 28 and 91 per cent. In the majority of instances over 60 per cent was recovered.

Other investigations led them to the belief that the kidneys could absorb 0.2 per cent of injected uric acid and because this was far below the average amount of unrecovered uric acid, they felt that most of this unrecovered portion must have been destroyed within the body.

They next applied the same tests to the patients suffering from well defined gout. At first they were afraid to give the intravenous uric acid in as large dosages as had been given to normal persons. This was later proved to be an unfounded fear, as in no instance was an attack produced. The results were again plotted (in the same figure) and a comparison is interesting. In the latter group the points of interest are:

(a) The endogenous excretion varied between 200 and 400 mg. per 24 hours.

(b) The amount of injected uric acid recovered in the urine varied between 15 and 40 per cent. In the majority of instances less than 25 per cent was recovered.

How to interpret their findings in cases of gout is difficult. The obvious explanation is either that there was increased blood retention, tissue absorption or uric acid destruction.

Folin and his collaborators have shown that except for the kidney but small amounts of uric acid may be taken up by the tissue. In the patients investigated the blood uric acid content remained above its control level for a longer period than in the normal cases. Furthermore, the uric acid level of the blood in gout was generally higher than normal. However, with these possibilities it is difficult not to conclude that there must have been increased destruction of uric acid.

On our group of cases purine metabolism tests were made and the results classified below. The patients were placed on a purine free diet for six to nine days. On one day, varying from the fourth to the sixth day, there was added to the diet one-half pound of calf's liver, which is equivalent to 200 to 230 milligrams of purine nitrogen, or 600 to 690 milligrams of uric acid. Total twenty-four hour specimens of urine were collected, and after mixing and measuring a portion of

each day's urine was kept in the ice box until the end of the test. At this time quantitative determination of uric acid was made by the method of Benedict and Franke.[6] Blood uric acid determinations were also made to follow the changes in dietary regimen; Benedict's method was used.[7]

It was found that the results of the test could be grouped and four types of curves plotted (*Fig. 10*). In *Lines a, b,* and *c,* the 24-hour endogenous excretion of uric acid was in the neighborhood of 350 mg. In *Line d* the endogenous output was much lower, approximately 200 mg., and furthermore, none of the ingested uric acid was recovered in the urine; all cases falling in the latter group were considered to have an abnormal purine metabolism. Analysis of the other groups shows that in *Line a* the control endogenous level was attained again in 48 hours. In four instances out of eighteen less than 25 per cent of the ingested uric acid was recovered and these four cases were considered to have an abnormal purine metabolism.

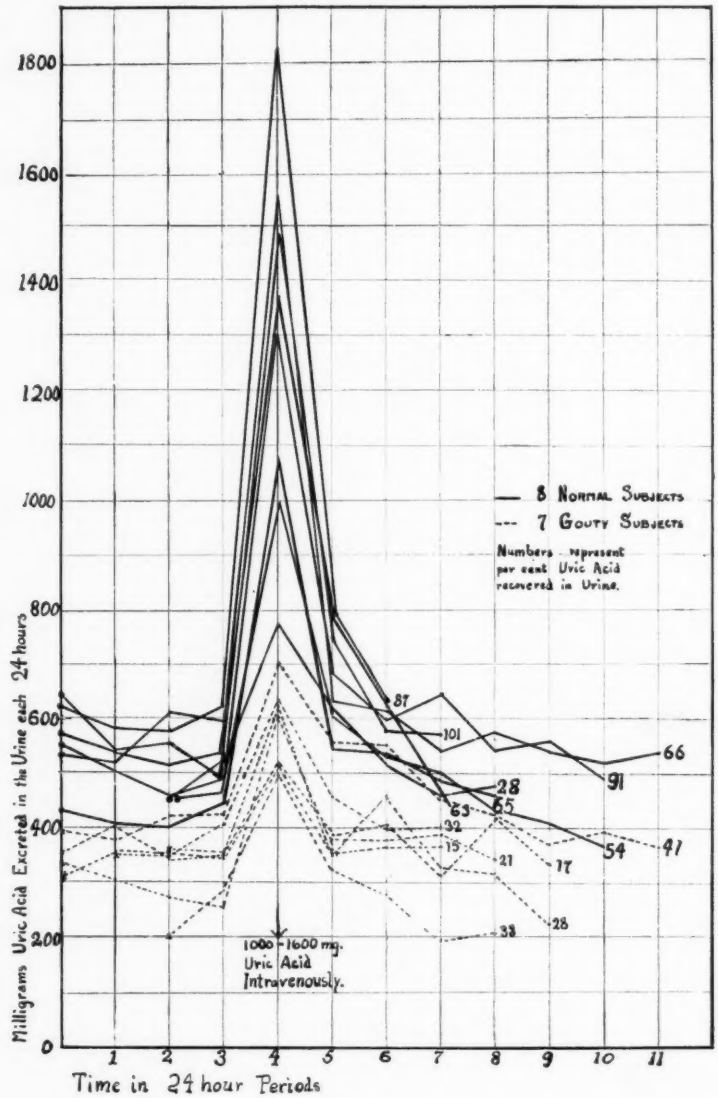

Fig. 9

Graph made from the tables in the article of Folin, Berglund and Derick showing urinary changes produced by the intravenous injection of uric acid in persons on a purine low diet.

Line b. The control endogenous level had not been reached at the end of 48 hours. In three instances out of fourteen less than 25 per cent of the ingested uric acid was recovered and these three cases were considered to have an abnormal purine metabolism.

Line c. There was a decrease of uric acid excreted on the day of purine ingestion. On the following day or two an increased amount was excreted. However, the total amount recovered, that is, using the endogenous level as control, was above 18 per cent only once. In several instances none was recovered. These cases were considered to have an abnormal purine metabolism. All the cases in this grouping had abnormal sugar tolerance curves.

Blood sugar estimations were made before and one-half, one, two and three hours after the administration of 100 grams of dextrose. Capillary blood was obtained by digito-puncture and blood sugar determined by the method of Folin.[8] The figures of Jaergaard have been taken as normal, that is, a rise over 190 mg. dextrose per 100 c. c. of blood or a curve which has not returned to the fasting level in three hours is accepted as abnormal.

The basal metabolic readings were made on a Benedict - Roth machine and the Boothby and

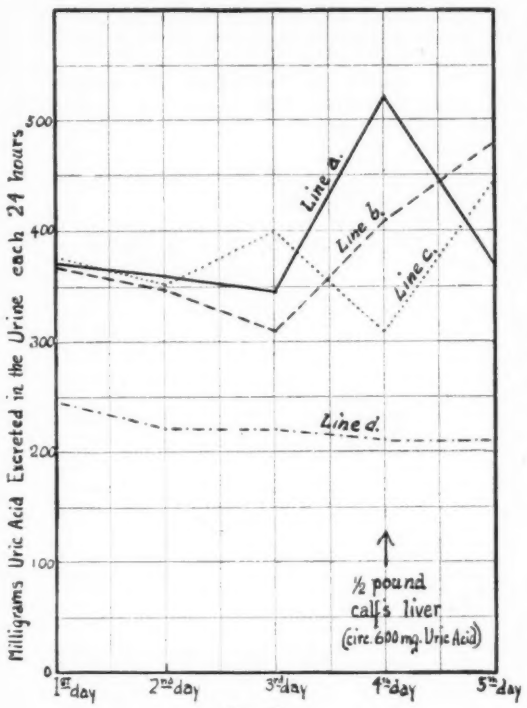

Fig. 10

Graph illustrating the various types of urinary response to high purine oral administration. Each line is the average of the total number of cases in that particular grouping (consult text and tables).

Sandiford[9] modification of the Aub-DuBois standards were used in the calculation of the basal rates.

Gastric analysis was made by the fractional method after stimulation with alcohol or histamine. Dimethyl aminobenzaldehyde and phenolphthalein were used as indicators in titrating.

It was felt that 9 of these cases fell within the infectious group, not only because of the fact that some harbored localized areas of infection which may have been possible foci but because of the clinical course of the disease. The data for these cases may be seen in *Table I.*

Two cases had a lowered sugar tolerance; two out of the seven upon whom purine metabolism determinations were made showed some evidence of delay in excreting exogenous uric acid. In four of the cases there was a lowering of the basal metabolic rate, but only markedly so in two. Great care was paid to the fluoroscopic examination of the colon and in no instance was a condition found which, from an x-ray point of view, could indicate the probability of an intestinal focus of infection.

The second lot of cases, *Table II,* comprises 12 patients whose group it has been impossible to determine with any degree of assurance. In 7 patients there was no abnormality in sugar or purine metabolism. Clinical findings were such that no clear-cut picture could be made out and, although hypertrophic changes were demonstrated in the x-ray films, it was not felt that in connection with other findings these cases could be placed in the metabolic group.

Forty-three cases selected by history, physical examination, x-ray and laboratory studies fell into the metabolic group, *Table III.* The onset was usually insidious; generally one joint became affected with a later distribution into two or more joints. The diseased joints were rarely inflamed; there was usually little swelling, but almost always crepitation on movement. The majority of the patients were beyond middle age and usually well nourished.

With the exception of 4 cases, x-ray films showed bony changes within the joint cavity: anything from small spicules at tendon insertions and slight lipping of the vertebrae to marked spurs and deposits along the cartilage-epiphyseal junction. As mentioned before, there was no direct connection between the severity of symptoms and the amount of bony deposit.

Metabolic studies have demonstrated some interesting changes. In 50 per cent of the patients there was a lowering of sugar tolerance; in 40 per cent, there was an abnormality in purine metabolism; and in 30 per cent, there was an abnormality in both.

Most of the basal metabolic rates were normal; in only eight instances was there a marked divergence from the accepted standard and in each of these the rate was below, 20 per cent.

Eighty per cent of the patients on whom gastric analyses were made demonstrated the presence of free hydrochloric acid in the gastric juice.

Fluoroscopic examination of the colon over a period of several days demonstrated no type of abnormality either in position, tone, shape or motility which might be associated with arthritis. It was necessary to regard practically all of them as normal colons and of the type to be expected in relation to the build, age and abdominal muscular tone of the particular patient.

With the method used for a number of years in the determination of blood uric acid, 3.5 to 4.5 mg. per 100 c. c. of blood has been accepted as normal. A glance at the table shows that the majority of determinations were found to be somewhat higher and some of them markedly so. A few series of blood uric acid determinations were made in connection with the changes in dietary purine intake. In these few cases it was possible to demonstrate that after a high purine feeding the blood uric acid became slightly elevated.

Table I*

CASES OF ARTHRITIS CLASSED AS INFECTIOUS

Case No. and Age	Sugar Tolerance Test. Time, Hours	Mg. Per Cent	Purine Metabolism Mg. Uric Acid	Fig. 10, *Line*	Basal Rate Per Cent	Blood Uric Acid Mg. Per Cent	X-Ray Joints	Treatment	Results
7—33 yrs.	C. ½ 1 1½	75 130 .. 144	281 251 331 385 (liver) 314 Exc. 25%	*a*	..	4.5	No changes	Dietary, then vaccine	Improved
12—43 yrs.	C. ½ 1 1½	100 138 .. 121	399 157 316 (liver) 296 300 Exc. 50%		—18	..	No changes	Dietary Low purine Low carbohydrate	
13—19 yrs.	C. ½ 1 1½	96 106 .. 104	..		—11	..	Large spur on upper side of Achilles	Surgery	Cured
19—36 yrs.	C. ½ 1 2 3	90 .. 210 200 87	189 167 184 138 (liver) 197 Exc. 2%	*d*	—34 —23	2.6	Infectious arthritis of fingers	Dietary; low carbohydrate	Better
24—23 yrs.	C. ½ 1 1½	84 110 .. 95	210 388 348 454 (liver) 460 Exc. 44%	*b*	— 7	..	Obliterated sacroiliac	Dietary Low purine	
27—56 yrs.	C. ½ 1 1½	100 118 .. 114	550 230 300 390 (liver) 440 Exc. 33%	*b*	—23	4.2	Spur on patella	Dietary Vaccine	No change
46—52 yrs.	C. ½ 1 2 3	81 200 182 143 129	..		—17	.. 4.5	No changes	Surgery Low carbohydrate	Cured
54—30 yrs.	C. ½ 1 2 3	100 .. 166 153 112	330 355 335 349 (liver) 516 540 Exc. 61%	*b*	+ 1	4.5	No changes	Dietary Vaccine	No improvement
55—51 yrs.	C. ½ 1 2 3	111 133 129 108 95	225 225 285 300 432 (liver) 225 295 220 Exc. 27%	*a*	± 1	4.0	No changes	Dietary Vaccine Drainage of sinuses	Improved

Note: Exc. per cent gives per cent of ingested uric acid (600 mg.) recovered (excreted) in the urine.

*For lack of space data relative to gastric acidity, blood calcium and phosphorus, and x-rays of colon and gallbladder have been omitted.

Table II*

CASES OF ARTHRITIS OF DOUBTFUL ETIOLOGY

Case No. and Age	Sugar Tolerance Test. Time, Hours	Mg. Per Cent	Purine Metabolism Mg. Uric Acid Fig. 10, *Line*	Basal Rate Per Cent	Blood Uric Acid Mg. Per Cent	X-Ray Joints	Treatment	Results
3—50 yrs.	C. ½ 1 2 3	80 182 143 149 93	.	—15	..	..	Dietary Low carbohydrate	Better
16—30 yrs.	C. ½ 1 1½	95 98 .. 95	672 712 .. (liver) 1313 1313 Exc. 100%	— 5	4.7	Hypertrophic changes in shoulder	Dietary Low purine Low carbohydrate	Slight improvement
18—52 yrs.	C. ½ 1 1½	116 .. 133 94	370 *b* 225 328 (liver) 410 Exc. 33%	—15	5.3	..	Dietary Low starch Anti-biliary	Improved
23—51 yrs.	C. ½ 1 1½	95 151 .. 100	260 *a* 270 280 390 (liver) 280 Exc. 23%	—16	3.1	..	Cholecystectomy	Improved for a while
26—48 yrs.	C. ½ 1 1½	95 174 .. 125	310 *a* 360 485 (liver) 365 Exc. 28%	—18	4.3	..	Dietary Low purine	..
34—49 yrs.	C. ½ 1 1½	100 121 .. 90	396 478 465 620 (liver) Exc. 26%	—11	..	..	..	..
43—62 yrs.	C. ½ 1 2 3	115 250 205 181 95	..	..	..	..	Dietary Low carbohydrate	Improved
45—50 yrs.	C. ½ 1 2 3	95 121 111 108 105	* 367 *a* 276 282 ** 566 (liver) 714 (liver) 393 Exc. 65%	—14	*4.4 **5.0 5.0	Lipping of cervical vertebrae	Dietary Low carbohydrate	Improved
47—50 yrs.	C. ½ 1 2 3	95 156 133 125 69	364 *b* 345 288 265 389 (liver) 470 425 430 Exc. 70%	..	4.2	No changes	Appendectomy	Not improved
49—42 yrs.	C. ½ 1 2 3	80 138 117 128 79	410 378 590 520 (liver) 560 (liver) 366 400 Exc. 7%	..	5.6	Lipping of one vertebra	Dietary Low purine Low carbohydrate	Cured
58—34 yrs.	C. ½ 1 2 3	117 190 167 112 81	240 *b* 268 278 180 294 (liver) 348 338 Exc. 38%	—13	..	Hypertrophic spurs on hand and feet	Dietary Low purine Low carbohydrate	Improved
61—44 yrs.	C. ½ 1 2 3	95 148 132 132 118	* 250 *d* 275 285 (liver) ** 371 270 288 *** 310 Exc. 20%	+ 1	*4.0 **4.5 ***4.5	..	Dietary Low purine	Improved

Note: Exc. per cent gives the per cent of ingested uric acid (600 mg.) recovered (excreted) in the urine.

*For lack of space data relative to gastric acidity, blood calcium and phosphorus, and x-rays of colon and gallbladder have been omitted.

Table III*

CASES OF ARTHRITIS OF METABOLIC ORIGIN

Case No. and Age	Sugar Tolerance Test. Time, Hours	Mg. Per Cent	Purine Metabolism Mg. Uric Acid	Fig. 10, *Line*	Basal Rate Per Cent	Blood Uric Acid Mg. Per Cent	X-Ray Joints	Treatment	Results
2—53 yrs.	C. ½ 1 2 3	110 125 100 95 100	946 863 943 876 (liver) 846 Exc. 0.		..	3.6	..	Dietary Low purine Low carbohydrate	No improvement Improvement
4—59 yrs.	C. ½ 1 1½	110 153 .. 142	..		..	4.1	Marked lipping of acetabulum with areas of rarification	Dietary Low carbohydrate	Improved
5—45 yrs.	C. ½ 1 1½	98 145 .. 110	..		—37	..	Deposits about sacro-iliac	Dietary Low carbohydrate	Improved
6—35 yrs.		..	435 164 158 (liver) 126 Exc. 0.	*d*	— 2	4.2	Spicules on knee	Dietary Low carbohydrate and low purine	Cured
8—55 yrs.	C. ½ 1 2 3	101 225 220 186 165	433 402 395 400 550 (liver) 460 Exc. 31%	*a*	—25	4.2	No changes	Dietary Low carbohydrate Thyroid extract	Cured
9—70 yrs.	C. ½ 1 1½	100 200 .. 143	454 386 430 472 (liver) 520 Exc. 22 %	*b*	+ 6	..	Spicules on knee	Dietary Low carbohydrate Low purine	No improvement
10—34 yrs.	C. ½ 1 1½	106 133 .. 177	348 350 468 (liver) 390 331 Exc. 25%	*a*	± 1	3.2	Spiculation of ligaments	Dietary Low carbohydrate	Improved
11—48 yrs.	C. ½ 1 1½	105 162 .. 115	385 377 409 (liver) 501 Exc. 33%	*b*	—21	3.8	No changes	Dietary Low carbohydrate	Cured
14—52 yrs.	C. ½ 1 1½	100 153 ... 142	273 268 161 449 (liver) 314 Exc. 40%	*a*	+ 2	5.2	Spinous process along great toe	Dietary Low carbohydrate	Improved
15—55 yrs.	C. ½ 1 1½	119 155 .. 125	418 387 296 542 (liver) 496 Exc. 60%	*a*	—12	..	..	Dietary Low carbohydrate and low purine	No improvement
17—45 yrs.	C. ½ 1 1½	83 250 .. 125	263 272 265 295 (liver) 232 Exc. 4%	*d*	—21	..	Lipping of vertebra	Dietary Low carbohydrate	No improvement
20—54 yrs.	C. ½ 1 1½	147 308 .. 313	..		..	..	Hypertrophic changes	Dietary Low carbohydrate	Died with cancer of pancreas
21—59 yrs.	C. ½ 1 1½	89 118 .. 116	696 493 456 600 (liver) 540 Exc. 42%	*a*	± 1	4.2	Marked lipping of acetabulum with degenerative changes	Dietary Low purine Low carbohydrate	No improvement

*For lack of space data relative to gastric acidity, blood calcium and phosphorus, and x-rays of colon and gallbladder have been omitted.

Table III—(Continued)

Case No. and Age	Sugar Tolerance Test. Time, Hours	Mg. Per Cent	Purine Metabolism Mg. Uric Acid	Fig. 10, *Line*	Basal Rate Per Cent	Blood Uric Acid Mg. Per Cent	X-Ray Joints	Treatment	Results
22—43 yrs.	C. ½ 1 1½	80 118 .. 93	290 455 355 475 (liver) 340 Exc. 18%	*a*	+ 1	3.0	..	**Dietary** **Low purine and low carbohydrate**	
25—40 yrs.	C. ½ 1 1½	105 221 .. 148	364 215 270 200 (liver) 273 Exc. 0.	*c*	— 1	5.5	No changes	Dietary Low purine	Cured
28—47 yrs.	C. ½ 1 2 3	111 200 160 148 76	*293 240 383 336 (liver) 415 Exc. 18%	*c*	—13 — 9	*5.3 3.8	Spicules in cervical vertebra and shoulder	Dietary Low purine and low carbohydrate	Improved
29—63 yrs.	C. ½ 1 1½	91 120 .. 98	285 285 575 (liver) 625 Exc. 30%	*b*	—20	4.4	Marked lipping of acetabulum with areas of rarification	Dietary Carbohydrate low	Improved
30—43 yrs.	C. ½ 1 1½	85 89 118 125	585 430 590 630 (liver) 515 Exc. 19%	*a*	— 5	5.9	Bony deposits between toes	Dietary Low purine and low carbohydrate	Improved
31—42 yrs.	C. ½ 1 2 3	95 185 215 195 160	465 440 435 470 (liver) 600 Exc. 23%	*b*	+ 4	..	..	Dietary Carbohydrate low	Cured
32—54 yrs.	C. ½ 1 1½	74 147 .. 105	430 434 519 555 (liver) 519 Exc. 20%	*a*	— 8	4.1	..	Dietary Low purine	Improved
33—30 yrs.	C. ½ 1 2 3	93 121 200 166 114	323 270 395 (liver) 400 430 Exc. 40%	*b*	—20	5.0	Hypertrophic deposits	Dietary Carbohydrate low	Improved
35—56 yrs.	C. ½ 1 2 3	100 240 174 222 174	495 518 450 (liver) 515 Exc. 0	*c*	— 5	4.3	Spicules at crurals and patella	Dietary Low carbohydrate	Improved
36—41 yrs.	C. ½ 1 1½	94 170 .. 174	..		—15	..	..	Dietary Carbohydrate low	Improved
37—62 yrs.	C. ½ 1 2 3	97 174 133 133 117	350 240 263 243 (liver) 334 Exc. 0	*c*	+ .3	..	Marked deposits about knee	Vaccine Low purine	..
38—56 yrs.	C. ½ 1 2 3	107 200 181 142 111	*418 378 425 335 (liver) **400 Exc. 0	*c*	+ 5	*5.7 **4.0	Marked deposits about knee	Dietary Low purine and low carbohydrate	Cured
39—48 yrs.	C. ½ 1 2 3	100 135 129 105 80	638 510 440 *560 437 (liver) 510 (liver) 646 (liver) **700 (liver) Exc. 0		— 7	.. *5.2 **9.4	Deposit on spine	Dietary Low purine	..

Table III—(Continued)

Case No. and Age	Sugar Tolerance Test. Time, Hours	Mg. Per Cent	Purine Metabolism Mg. Uric Acid	Fig. 10. *Line*	Basal Rate Per Cent	Blood Uric Acid Mg. Per Cent	X-Ray Joints	Treatment	Results
40—60 yrs.	C.	92	298	*a*	—26	6.0	Hypertrophic deposits	Dietary Low purine and low carbohydrate	Improved
	½	112	443						
	1	160	390						
	2	143	*466 (liver)			*5.4			
	3	148	534 (liver)						
			**376			**6.0			
			367 Exc. 40%			6.3			
41—40 yrs.	C.	121	350	*a*	— 2	5.0	No changes	Dietary Low carbohydrate	..
	½	250	250						
	1	250	175						
	2	250	*250			*5.0			
	3	125	296						
			**444 (liver)			**5.0			
			349						
			***279 Exc. 41%			***5.7			
42—50 yrs.	C.	108	*370	*a*	—22	*4.8	..	Dietary Carbohydrate low	Improved
	½	160	536						
	1	167	258						
	2	123	472 (liver)						
	3	153	256 Exc. 25%						
			**			**4.4			
44—44 yrs.	C.	87	435	*a*	— 6	4.0	Marked lipping of acetabulum with areas of rarification	Dietary Various— Acid-ash—	Not improved ?
	¼	154	365						
	1	138	237						
	2	111	522 (liver)						
	3	77	790 (liver)						
			516						
			374 Exc. 63%						
48—33 yrs.	C.	91	464		— 7	4.0	..	Dietary	..
	½	134	344						
	1	91	325						
	2	111	470 (liver)						
	3	85	236						
			279 Exc. 14%						
50—60 yrs.	C.	100	313	*c*	—27	4.7	Very small osteophytes about tibia, femur and patella	Dietary Carbohydrate low	Improved
	½	143	290						
	1	200	334						
	2	200	317 (liver)						
	3	148	407 Exc. 18%						
51—45 yrs.	C.	111	*458	*b*	— 6	*4.7	Bony deposits	Dietary Low purine and low carbohydrate	Better
	½	174	380						
	1	143	**420			**4.0			
	2	137	***466 (liver)			***4.7			
	3	80	566						
			370 Exc. 18%						
52—73 yrs.	C.	129	258	*b*	+ 6	4.1	Spicules at attachment of ligaments	Dietary Carbohydrate low	Cured
	½	173	328						
	1	182	*466			*4.1			
	2	200	202						
	3	173	376 (liver)						
			**464			**4.7			
			452 Exc. 53%						
53—42 yrs.	C.	100	173		+ 1	..	..	Dietary	..
	½	138	..						
	1	182	*245			*5.2			
	2	133	196						
	3	69	300 (liver)						
			**278 Exc. 23%			**4.7			
56—39 yrs.	C.	143	*512	*c*	—11	*4.7	Slight lipping of cuboid bone	Dietary Carbohydrate low	Improved
	½	173	515						
	1	182	595						
	2	148	**319 (liver)			**5.7			
	3	118	755						
			545 Exc. 34%						

Table III—(Concluded)

Case No. and Age	Sugar Tolerance Test. Time, Hours	Mg. Per Cent	Purine Metabolism Mg. Uric Acid	Fig. 10, *Line*	Basal Rate Per Cent	Blood Uric Acid Mg. Per Cent	X-Ray Joints	Treatment	Results
57—53 yrs.	C.	91	425		+ 5	4.7	Hypertrophic changes in fingers	Dietary Carbohydrate low	Improved
	½	138	455						
	1	129	435						
	2	94	450 (liver)						
	3	83	352						
			348 Exc. 1%						
59—50 yrs.	C.	110	*280	*b*	—26	*4.7	Hypertrophic changes	Dietary Carbohydrate low Thyroid extract	Not improved
	½	183	258						
	1	167	180						
	2	167	284						
	3	167	372 (liver)						
			**390			**4.0			
			370 Exc. 47%						
60—51 yrs.	C.	100	242		— 2	4.0	Marked hypertrophic deposits along spine	Dietary Carbohydrate low and purine low	Not improved
	½	200	272						
	1	200	284						
	2	147	245 (liver)						
	3	138	356						
			381						
			308 Exc. 42%						
62—39 yrs.	C.	95	197	*d*	± 1	..	..	Dietary Low purine and low carbohydrate	Marked improvement
	½	160	192						
	1	160	255						
	2	125	200 (liver)						
	3	90	*210			*4.7			
			205						
			210 Exc. 4%						
63—58 yrs.	C.	110	128	*d*	+ 1	..	Hypertrophic deposits	Dietary Low purine and low carbohydrate	Improved
	½	222	135						
	1	235	147						
	2	117	208 (liver)						
	3	83	190						
			166 Exc. 28%						
64—61 yrs.	C.	92	325		+ 1	..	Hypertrophic changes		Improved
	½	160	310						
	1	165	*310			*3.2			
	2	105	**339 (liver)			**4.0			
	3	100	208						
			***183 Exc. 3%			***4.0			
65—62 yrs.	C.	112	*262	*a*	+ 3	*3.2	Marked hypertrophic changes in knee	Dietary Low purine and low carbohydrate	No improvement
	½	190	379						
	1	136	351			**2.7			
	2	92	**224						
	3	104	504 (liver)			***2.7			
			***280 Exc. 31%						

Note: Exc. per cent gives per cent of ingested uric acid (600 mg.) recovered (excreted) in the urine.

DISCUSSION

It is interesting and perhaps important that in those cases classified as infectious but two were found to have any disturbance in carbohydrate tolerance and only one with an abnormality in purine metabolism. In 6 of these cases we have repeatedly tried to demonstrate a streptococcus by the method of Cecil.[10] We have been as unsuccessful here as in the many attempts in cases in the metabolic group. Perhaps our technic has been at fault, but the instructions were rigorously observed. In 3 patients of the infectious group, an hemolytic staphylococcus, at first growing only on ascitic fluid media, was isolated and has been used as a vaccine with some improvement. One case was completely cured by excision of a Bartholin's gland cyst. The others have rocked along on non-specific vaccines.

A consideration of those cases listed in *Table II* of doubtful etiology shows no remarkable therapeutic results. Truly the greater number of them say they are better, but they all still suffer to a certain degree. They were all placed on either low carbohydrate or low purine diets, and it may be that a stricter adherence to regi-

men would have produced better results. It is fair to say that if these cases had shown more improvement, one would have been tempted to place them in the metabolic group. Classification has been influenced and simplified by the therapeutic results.

It is in the group of metabolic or osteo-arthritic patients that the main interest centers. Their diet was regulated in relation and according to the type of metabolic abnormality; cases with a low sugar tolerance were placed on a sugar-free, starch low diet; cases with purine disturbance on a purine low diet, and the 12 cases with combined findings have had to suffer a doubly restricted regimen. When no carbohydrate or purine abnormalities were found the patients were tried first on a carbohydrate low, and if that did not succeed, then on a purine low or combined diet.

A study of the results of the different types of treatment shows:

(1) Of the cases having a low sugar tolerance, 70.5 per cent are clinically cured on a carbohydrate low diet.

(2) Of the cases having an abnormal purine metabolism and a normal sugar tolerance, 66.0 per cent are clinically cured and all but one improved. Diet was adjusted as follows: carbohydrate low, 33.3 per cent; purine low, 25 per cent; combined purine and carbohydrate low, 41.7 per cent.

(3) Of the 13 patients having both abnormal sugar tolerance and abnormal purine metabolism, 76.9 per cent are clinically cured.

(4) Of the 4 cases having a normal sugar tolerance and a normal purine metabolism, 3 are unimproved.

In brief, 60.6 per cent of the cases of metabolic arthritis are now without symptoms. A certain number, 21.2 per cent, admit that they have been improved at one time and some blame their lack of better results on dietary temporizing. Eighteen and two-tenths per cent are thus without improvement of any kind even though treatment has been rigorously followed.

As has been noted before, 8 cases had a basal metabolism lower than —20 per cent; all of these cases were given thyroid extract in addition to their dietary readjustments. Six were among the most successfully treated patients.

COMMENT

It is difficult to decide how to classify a large number of cases; whether they fall into the infectious or metabolic type of arthritis. There are extreme cases of both types which are easily classified, but patients of about middle age require long study to establish a diagnosis; and in this investigation it was impossible to classify satisfactorily 20.3 per cent even with long study.

Naturally the question will arise as to whether one is justified in classifying those cases as metabolic because of certain indefinite abnormal history, physical, laboratory and x-ray findings even in spite of the fact that when observing certain dietary restrictions improvement is noted. It might be argued that the change in diet in some way affects a sensitized area or neutralizes the toxins or antigens which were previously activating it. The pathological degenerative findings of these cases must be kept in mind, although no infection or inflammation is suspected. The x-ray findings in the present light of knowledge can be more intelligently explained by degenerative changes.

Other matters of real interest arise. How is it possible to explain the absence of symptoms in those joints which after treatment show the same amount of bony deposit as in the control films? What has the diminution of the carbohydrate or purine intake or both done to lessen the irritation of the intra-articular tissues by an unknown toxic substance?

SUMMARY

(1) Data obtained from the study of 65 cases has been given under three groups:

(a) Those persons having infectious arthritis.

(b) Those persons having metabolic arthritis.

(c) Those persons in which it was impossible to designate the type.

(2) Classification was made on the basis of history, physical examination, x-ray studies of affected joints, estimation of purine metabolism and carbohydrate tolerance.

(3) The criteria for the classification of a case have been given.

(4) Treatment has been based on the results of the clinical and the laboratory studies.

(a) The infectious type of case was treated by general upbuilding measures; removal of foci of infection; vaccines, autogenous and stock; rest.

(b) The metabolic type of case was treated mainly on a dietetic basis:

(1) Seventy and five-tenths per cent of the cases with a low sugar tolerance were clinically cured while on a carbohydrate low diet.

(2) Sixty-six and six-tenths per cent of the cases with an abnormal purine metabolism were clinically cured and all but one improved; a quarter were on a purine low diet, a third on a carbohydrate low and the rest on a combined low diet.

(3) Seventy-six and nine-tenths per cent of the cases with both a low sugar tolerance and an abnormal purine metabolism are clinically cured.

(4) Only one of the cases with both normal sugar tolerance and normal purine metabolism is improved.

(5) Altogether, 60.6 per cent of the cases with a metabolic type of arthritis are now clinically cured.

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ENDEMIC NUTRITIONAL EDEMA IN TENNESSEE: A PUBLIC HEALTH PROBLEM*

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Among the diseases which are of importance to public health are certain disorders of nutrition. Unlike the infectious diseases, the individual cases offer no menace to the health of others except indirectly and remotely. They become of public health interest when they are the result of conditions which are common to any considerable part of a population rather than peculiar to the individual. From a broad point of view the most common is general undernutrition, the effect of which, on individual and public health can be determined only very imperfectly at present. Among the more specific disorders of nutrition such diseases as pellagra, beriberi, rickets and simple goiter have long been considered problems of public health interest.

The association of edema with undernutrition has been recognized for centuries. Since the World War its relation to an inadequate supply of protein and a hypoproteinemia has been clearly demonstrated.[1] Emphasis has been placed, however, on its appearance in epidemics, usually in association with such calamities as war and famine, or as sporadic cases in individuals who are malnourished as the result of some disease. In this country it has been the sporadic cases which have attracted attention.

For the past four years my associates and I have been observing and studying cases of endemic edema which we believe to be nutritional in nature.[2] Though endemic, the disease has shown a seasonal variation in incidence, the majority of the cases appearing in the late winter, spring and early summer. Adults of all ages are affected* and women more often than men. The edema has been mild to moderate in degree in the majority, involving the feet and legs most frequently, but sometimes the face and hands as well. The duration has varied from a few days to several months, but many have given a history of previous attacks and in a few we have been able to observe annual attacks over a period of several years. Some of the patients have complained of mild pain and tenderness in the affected parts, but none have shown definite evidence of peripheral nerve changes. A purpura has appeared on the legs of three or four. In accord with the rather mild nature of the edema, most of the patients have not been severely undernourished. Many have had a slight or moderate secondary anemia.

In spite of the absence of any special or unusual causes for a general undernutrition the edema was suspected of being nutritional in nature. Therefore, studies were made of the diets and serum proteins of these patients. The study of the diet showed that in nearly every case the total calories were close to or below the basal requirements of the individual. The protein intake was below the usually accepted minimum of 0.6 gm. per kilogram of body weight in many instances, and in practically all it was below 1 gm. per kilogram. *Chart 1* shows the average total calory and protein intake of 13 of these patients who have been seen recently. For comparison there is presented an estimate of the average total calory and protein requirements of the same group. It should be emphasized that

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*A similar condition has been observed in children.

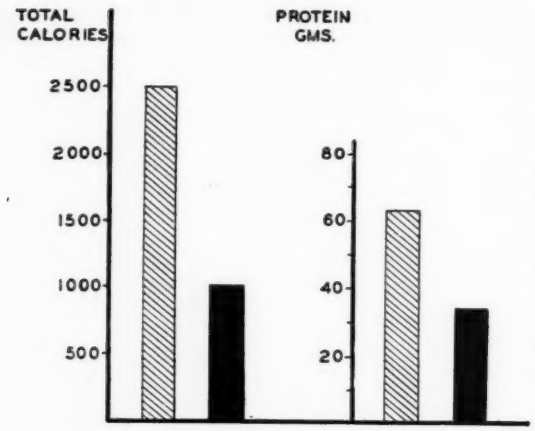

Chart 1

A comparison of the average total calory and protein intake per day of 13 patients with nutritional edema with an estimate of the average total calory and protein requirements. The latter are not considered optimal values. Solid columns represent the average intake; shaded columns the estimated requirements.

the latter are only approximate and are not believed to constitute the optimal amounts of these food factors. They are presented merely for a rough comparison.

Certain other features of these diets are of interest. Most of the diets are exceedingly monotonous and unappetizing and the preparation of the food is often faulty. A sample diet is shown in *Table 1*. Many of them are probably inadequate in other respects than total calories and protein, and in general the diets are of a kind which might cause at least mild forms of pellagra, beriberi and scurvy. In view of our ignorance of the subclinical and latent forms of these diseases, such a possibility must be considered. Frank pellagra does in fact occur among the same group of the population from which come the cases of edema. It is also possible that a minimal or subminimal intake of vitamin B_1 may lead to a decreased appetite with

TABLE 1

FOOD INTAKE* FOR ONE WEEK OF A PATIENT WITH NUTRITIONAL EDEMA

Breakfast

Food	Days	1st	2nd	3rd	4th	5th	6th	7th
Biscuits (1)		+	+	+	+	+	+	+ (2)
Coffee		+		+	+	+	+	+
Eggs (1)			+			+		+ (2)
Pork sausage (1)					+			
Fried apples				+				
Peach butter							+	
Butter				+				

Composite breakfast: 1 cup of coffee, 1 biscuit, 1 egg or 1 sausage. Occasional bit of preserves.

Dinner

Corn bread		+	+	+	+	+		+
String beans		+		+	+			
Irish potatoes				+		+		
Steak			+				+	
Buttermilk		+	+					
Biscuit (1)						+	+ (2)	
Milk					+			
Fried apples						+		
Liver							+	

Composite dinner: Corn bread, stringbeans or potatoes, small piece of meat, glass of buttermilk or milk.

Supper

Corn bread		+		+	+		+	
Buttermilk		+		+			+	
Crackers (2)			+					+
Cheese			+					+
Milk					+			
Cake						+		

Composite supper: Cornbread, buttermilk. Occasional crackers and cheese.

*Unless otherwise indicated, the amounts are a small piece or serving, or, with liquids, one cup or glass. In this instance the average daily calories are 790 and the protein 31 grams.

a further decrease in food consumption and the establishment of a vicious circle. All of this emphasizes the fact that in the clinic deficient diets are usually deficient in more than one respect, and illustrates the difficulty of studying the effect of single dietary deficiencies in patients.

These diets assume a greater importance when it is realized that in most instances they have been followed for years and in many cases for most of the patients' lives. This is particularly significant with respect to the possibility of disorders arising from the long-continued use of minimal or suboptimal diets. These diets are probably more the result of custom and habit than poverty. Inquiry has revealed that the diet is not only considered adequate, but is preferred to others by many of the patients. In some instances, however, economic causes are responsible. How much the current beliefs with respect to the protein requirements have influenced these dietaries is questionable, but they probably have had little effect. Although most of these patients are from urban communities, the dietary habits and customs appear to be a carry-over from a rural life in which the diet was the result of certain fundamental socio-economic conditions unrelated to scientific beliefs regarding diet.

In the majority of the cases there was a slight to moderate reduction in the serum albumin, though the total proteins were usually within the normal range due to a high normal, or higher than normal, globulin. Since albumin has an osmotic pressure approximately four times that of globulin, the reduction in the former seemed significant. These findings are illustrated in *Chart 2.*

In those patients who could be followed a sufficient time there was exhibited a tendency for the edema to disappear as the serum proteins increased, but this relationship was not as close as might be expected, probably because of the influence of various secondary factors. From a practical point of view, it was difficult to increase the protein intake sufficiently to bring about a prompt rise in the serum proteins, and even when a large intake of protein was secured a considerable time was necessary before the serum proteins rose to their maximum values. It is unknown whether abnormalities in absorption or utilization may have resulted from the long-continued subminimal intake.

The possibility that the edema is the result of the lack of some specific factor, an avitaminosis, demanded consideration. On the basis of the clinical features beriberi seemed the only known disease with which the condition might be confused, though the possibility that some as yet unknown factor might be concerned still remains. The administration of vitamin B_1 and B_2 (G) in the form of "vitavose" and brewer's yeast, respectively, and more recently the use of a much more potent B_1 concentrate, has failed to provide evidence that the edema is in any way directly concerned with these two factors. Analyses of the edema fluid do not indicate that the edema is the result of a capillary injury such as occurs in scurvy.

On this evidence we believe the disease to be a mild nutritional edema due to the long-continued use of a diet usually insufficient or barely sufficient in total calories and containing a minimum or less of protein. It probably differs only in degree from the more severe nutritional edemas which have been described previously. Just what relation it bears to the edema which may accompany certain other deficiency diseases as pellagra, beriberi or the epidemic dropsy of India is not entirely clear, though the diet in many of these latter diseases is one which might well lead to a limited intake of protein. Conversely, as we have seen, the diets of our patients are not so very different from those of some patients with beriberi and pellagra.

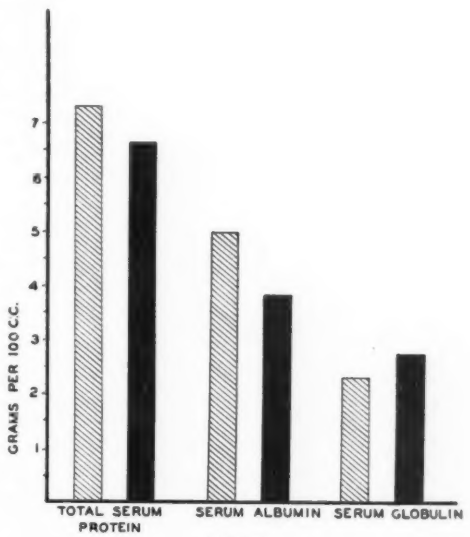

Chart 2

The average total serum protein, serum albumin and serum globulin values in 13 patients with nutritional edema compared with the average normal. The solid columns represent the patients' values, the shaded columns the average normal.

It should be emphasized that the edema in itself is probably of little importance. It is unlikely that it is, to any degree, injurious to the patient and it causes little disability. It is important mainly as an indication of a more fundamental disorder. Whatever may be the exact relationship between the serum proteins and the edema, a relationship which is modified by many other factors, the edema seems to be an indication of a chronic protein starvation and therein lies its importance. Unfortunately, there are no clearly recognized, proven signs and symptoms of a chronic, mild protein deficiency in man. Furthermore, we are ignorant of what constitutes the optimum amount of this important food substance and many of the disorders attributed to a lack of protein have been ascribed to other factors. Until very recently the majority opinion, or the most vocal opinion, has inclined to the belief that rather small amounts of protein are sufficient, if indeed, not preferable. However, insufficient attention has been paid to the factor of duration when interpreting the results of low protein diets,[3] with the possibility that untoward effects, which might result from the consumption of minimal amounts of protein over as long a time as in the case of some of these patients, may have been missed. Furthermore, the factor of safety has been lowered until insufficient allowance is made for complicating factors which might reduce the protein from a minimum to an inadequate amount. As far as can be judged from animal experiments,[4] the optimal amount of protein is probably much greater than these patients with edema have taken. Furthermore, the most recent studies seem to show that the larger amounts are not injurious even in conditions which might be expected to contraindicate their use.[5]

Although direct proof is lacking in man, a number of abnormal states have been attributed to an insufficiency of protein on the basis of animal experimentation and clinical observation. These include decreased resistance to disease, faulty development, lessened fertility, early senility, anemia, and decreased mental and bodily vigor. Some of these have been observed in persons subjected to more acute and severe protein deprivation and, to a lessened degree, might be expected to appear in the more mild and more chronic forms. Others, such as developmental defects, early senility, decreased resistance to disease, and sclerosis might be exaggerated. Granting that all this is true, the importance of a mild chronic protein starvation is very great. Among our patients edema was usually the principal symptom, but the long duration of other abnormal conditions, such as those described above, may have caused them to seem almost a normal condition to the patient. Anemia, which is the most objective finding, was very common. Lack of mental and bodily vigor, of energy and ambition, is a characteristic of these patients. The relative incidence of early senility and the degree of resistance to disease are difficult to determine, yet the impression persists that this group compares unfavorably in this respect with persons known to receive an adequate diet.

Matters of diet such as these cannot fail to be of importance to public health. Less likely to attract attention than the more severe cases due to gross inadequacies, the milder forms of dietary disease may be of greater fundamental importance. Less obvious, they remain unsuspected; their incidence is apt to be greater and their effects, including those which are remote and indirect, may be more serious. Granted that the effects of the dietary error which has been described include even part of the disorders which have been suggested, such a dietary fault must be important to public health wherever it is common.

The remedy for this situation is, first of all, education. These people must be taught what constitutes a proper diet. This means not only a knowledge of the proper amounts of the various food factors, but knowledge as to which of the available foods will supply them. To insure the actual consumption of these foods, the people must be taught in many cases how to prepare them and to like them. All of this may amount to a revolutionary change in long-established dietary habits and customs. In this program the part played by poverty must not be forgotten. It is to be hoped, however, the use of a correct diet will cause an improvement in mental and physical health, which, in turn, may raise the economic status of this part of the population. To accomplish these ends, more attention must be paid to diet in public health education.

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DISCUSSION (*Abstract*)

Dr. G. A. Wheeler, Washington, D. C.—The condition which Dr. Youmans describes has been encountered in connection with our epidemiological studies of pellagra, though not necessarily associated with that disease, in working with large groups of peop'e in the pellagrous sections of the South. The presence of an unexplained slight or moderate edema, or the history of a "touch of dropsy" the preceding spring or over a period of years, though not striking is at the same time not very unusual. Aside from the observation of its spontaneous occurrence, we have produced it accidentally on two occasions in connection with the experimental studies on pellagra and, in cooperation with Dr. N. P. Walker, Clinical Director, Milledgeville State Hospital, Milledgeville, Georgia, in a study of diet and epilepsy, a similar condition was produced intentionally in a group of epileptics. Its accidental production was in connection with tests of the pellagra preventive power of fresh beef and a dried watery extract of yeast. In both experiments the cornmeal and cowpeas in the basic diet were partially replaced by rice and pearl grits on the assumption that the beef and yeast extract supplements would provide sufficient antineuritic vitamin. Edema, beginning along the shins, appeared within a few weeks and was soon followed by tenderness over the calf muscles and tachycardia. Subsequent studies showed that the yeast extract used (a commercial preparation) was almost wholly lacking in the antineuritic vitamin and fresh beef much more deficient than we have been led to believe. In the intentional production of the condition in epileptics the disease began in the same manner and within a few weeks from its onset had, in several cases, advanced to such a marked degree that the experiment had to be discontinued. The diet used was the same as our regular pellagra-producing experimental diet with the exception that rice and grits replaced a portion of the cornmeal and cowpeas. Autoclaved yeast was added to protect against pellagra and purified casein as an additional source of protein.

The view that this condition is brought about by an inadequate supply of protein is not borne out under experimental conditions. The beef-supplemented diet was amply supplied with protein (1.5 gram per kilo) and the experimental diet given the group of epileptics was reinforced with protein of high biological value. Yet all of them came down with the condition quite promptly. On the other hand, the symptoms in all three instances cleared up under dietary changes which did not materially affect the protein level. In case of the beef and yeast extract diets, the condition disappeared following the replacement of the grits and rice with the customary proportion of whole cornmeal. In the epileptics the treatment consisted only of a substitution of p'ain dried yeast for the autoclaved yeast.

I like to think of this condition as a mild or larval form of beriberi. On this basis, the question of why we do not more often see well advanced and classical cases of beriberi might fairly be asked. This may possibly be explained by a high rate of spontaneous recovery as the growing season advances and a greater variety of foods, particularly green vegetables, become available in the rural sections of the South. It must be borne in mind that pellagra, in which disease the digestive system is impaired and spontaneous recovery rendered more difficult, at least 90 per cent of the endemic cases clear up under seasonal changes in food availability alone.

Dr. Youmans' observations serve to reinforce the urgent demands for more and better understood nutritional measures in pub ic health. The more our foods and foodstuffs are depleted of essentials by refining processes and the more general the carton, paper bag and can opener become in our kitchens, the more complete must be our knowledge of overcoming the dietary deficiency conditions which, in the absence of suitable supplements, will most surely follow. One way would be to work out a specific remedy to be applied for the correction of each particular fault but under prevailing conditions the blanket method would perhaps be more practicable and more effective. It takes time to determine the specific nature of a nutritional fault and work out a specific remedy, more time to convince the physicians and other health workers of its soundness, and still more time to educate the public as to what must be done to overcome the trouble. On the other hand, we know that a diet consisting of a reasonable variety of foods (not necessarily expensive) is a successful barrier against all known dietary deficiency disorders. If we can induce those (fortunately relatively few) among us who are not a'ready doing so, to provide and consume a more generous and a more continuous supply of vegetables and fruits of various kinds, poultry, and dairy products, or home grown meats, we need have no fear of pellagra, nutritional edema, or any of the other dietary deficiency disorders which are more and more undermining the health of a considerable element of the population of the rural South.

Dr. Henry E. Meleney, Nashville, Tenn.—The presentation of this interesting and important disease condition to the members of the Public Health Section illustrates the manner in which the clinician may be of assistance in pointing out to public health agencies conditions which may not be generally known to exist but which are of great importance to the health of the people. Dr. Youmans' contribution may be used by public health agencies in the education of the people of this section of the country not only toward improving their nutrition but toward getting away from improper food habits which have existed over a period of many generations and which have not been recognized as deleterious to health. His report also illustrates the value of using the out-patient department of well organized hospitals for the detection of disease conditions which may not come to attention in the hospital wards because of the apparent mildness of the disease. The cooperation of such institutions in pointing out public health problems and in their solution deserves the special attention of those who are working primarily for the public health.

Dr. Youmans (closing).—It is possible, as Dr. Wheeler has said, that the edema in these patients is due to the absence of some specific accessory factor, rather than to a deficiency of protein.

However, so far we have been unable to obtain evidence that this is the case and the only significant abnormalities we have been able to demonstrate are the changes in the serum protein. A close relationship between lowered serum protein and the formation of edema has been demonstrated in conditions similar to this and we are therefore inclined to look upon the serum protein changes in these cases as significant.

It is undoubtedly true that this form of nutritional edema occurs elsewhere than in Tennessee. I have, for example, received information recently that it is being observed at the present time in the Northern States. However, this paper deals only with those patients whom I have observed personally.

PEPTIC ULCER IN THE SOUTHEAST: A CLINICAL STUDY OF ONE HUNDRED AND EIGHTY CASES*

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One of the trends of internal medicine has been towards a greater recognition of the patient himself and of the individual to the disease process. We recognize more, too, the personality of the patient, which is colored by his disease, and whose disease in turn is tempered by his personality. We appreciate more the influences of environment and the variance it produces in certain pathological processes. Peptic ulcer is a disease entity, but also a process which is a part and product of the warp and woof of the patient himself.

The individual with peptic ulcer has long since learned to be a patient and tolerant host to his disease. From years of experience he knows the caprice of his guest.

The line of approach followed in this paper is a presentation of facts about the ulcer bearing individual and factors relating to the environment in which he lives. Two groups of cases are to be reported here. Controversial discussion of the theory and etiology of the disease will be avoided.

Recent statistics seem to offer differences in occurrence, in severity, in the ratio of gastric to duodenal ulcer and consequently a divergence of opinion as to its management, although in later years there is more agreement as to medical or surgical procedures.

Environmental and personality factors in the patient give rise to many of these differences and help to explain the variety of the symptoms both subjective and objective. It is for this reason that in this paper two groups were chosen for clinical study, namely: a white series and a colored series of peptic ulcer patients. The colored series is particularly useful for study, inasmuch as the two etiological and chronicity factors, those of focal infection and food habits, occur in a manner unparalleled in any race of people. The food habits of our people of the Southeast offer some facts for serious consideration, because of the close relationship of the ulcer problem and the patient's dietary.

The figures given below are primarily for the State of Georgia, but they also represent closely the situation in the other Southeastern states. In 1931, the State of Georgia spent approximately $100,000,000 for food products outside of the State. In the metropolitan area of Atlanta, a city of some 300,000 persons, out of 6,132 carloads of fruits and vegetables used, only 1,087 came from Georgia. We can grow as fine grades of such vegetables as beans, cabbage, cauliflower, carrots and spinach as in any other state, but we do not produce an adequate quantity of these products. Furthermore, we do not demand anywhere near the amounts of these foods to meet the normal dietary standards. The State College of Agriculture, in cooperation with the Georgia Experiment Station, has carried on studies of the diets of the rural people of this State, which are of sufficient importance to quote in detail. One hundred families in North Georgia and 100 families in South Georgia kept a record of all food consumed by their families for a period of two weeks during each of the four seasons of the year. All foods used were weighed and measured and a careful record was made of the sex and ages of all the persons who consumed the food. An average of the 800 dietaries (4 dietaries for each of the 200 families) revealed the fact that Georgia diets were adequate as regards calories, protein and calcium. There was a small deficiency of phosphorus and a very serious deficiency of iron. According to the accepted standards of nutrition, Georgia rural diets contained 45.2 per cent more fat than they should have. Bacon was included among the fats. There was an excess of 20 per cent of cereal food used. This included all breads, rice, grits and breakfast foods or cereals. Twenty-four per cent more sweets were used than was necessary. Sugar, syrup and honey were included in the group. Twenty-five per cent less meats, eggs

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and cheese were used than is recommended by a good standard of nutrition. Twenty and one-half per cent less milk was used. There was a deficiency of 48.2 per cent in the use of fruits and vegetables. To summarize, there were too many fats, starches and sweets used and too little milk, eggs, meats, fruits and vegetables. The scarcity of fruit and vegetables caused the deficiency of iron. There was not an excess of lean pork according to the best standards, but there was an excess of fat pork.

No effort was made to evaluate the vitamin content of the diet. It is evident, however, that there is a lack of vitamins, particularly B and C, due to the 48.2 per cent deficiency in fruits and vegetables. These findings are in accordance with similar studies, more extensive but less accurate, made by the U. S. Department of Agriculture. While no studies have been made of city dietaries in Georgia, studies in other states show that among other deficiencies the lack of calcium is a notable one.

Wilkerson, of Alabama, in a paper on "Diet Difficulties in the South" has called attention to some of our food defects, and Harris also has emphasized the avitaminized, sugar saturated individual, particularly in regard to the development of ulcer.

The two series of ulcer patients were studied as to age, sex, family history, duration of symptoms, focal infection, constipation, dietary history, chief symptoms, perforation, hemorrhage, hemoglobin values, blood pressures, gastric contents, roentgen appearance, associated disease, the history of tonsillar, appendical and gallbladder disease, hookworm disease, dentition, cases treated medically, cases treated surgically, occupation, and ultimate outcome.

In the white series the incidence of gastric to duodenal ulcer is 3 to 79. It is my belief that gastric ulcer is infrequent in the Southeast. Possibly the low residue diet and consequent lack of trauma to the gastric mucous membrane is responsible. In the colored series, which were hospital cases, a much higher incidence of gastric ulcer is found, namely: 23 gastric, 12 pyloric, and 65 duodenal ulcers. This may be explained by the vicious oral sepsis, the inadequate dentition and the unbalanced diet of the colored series.

Sex.—In the white series there were 76 males and 6 females; in the colored series 64 males and 36 females.

Age.—In the white series there was none under twenty years, the greatest incidence being between thirty and fifty. The youngest patient was twenty-three, the oldest seventy-three. In the colored series the greatest incidence was between twenty and forty. The youngest was fifteen. In a recently published paper, Foshee calls attention to the rarity of peptic ulcer in patients under fourteen years of age, and reports a case, bringing the total number of cases in the literature up to 19 in number.

Family History.—A familial or constitutional factor has long been associated with peptic ulcer. In the white series one patient had a brother with duodenal ulcer. One had a brother and two uncles. One had a father and sister with this disease; one had a mother and brother. There were also 4 cases of familial ulcer in the colored series.

Duration of Symptoms.—In the white series the greatest number had had symptoms from one to ten years. One had symptoms for 49 years, one for 44 years, one for 54 years. The duration of symptoms in the colored series was approximately the same.

Focal Infection.—In the white series abscesses at the roots of teeth were found in 37 cases, or nearly 50 per cent; in both teeth and tonsils in 9 cases; and tonsillar infection alone in 15 cases. Twenty cases had already had tonsillectomies. Thus there is some focal infection in each case in the entire group. In the colored series pyorrhea alveolaris, periapical abscesses and gross dental caries were found in all cases. The so-called perfect teeth of the ante-bellum negro is decidedly a thing of the past, and marked oral sepsis is the rule today among them. The extensive caries leads to extractions and consequent poor mastication. This results in varying degrees of gastritis, and accounts for the vomiting present in many negro ulcer patients. Tonsillar infection occurred in 22 per cent of these cases.

Chief Symptoms.—In the white series pain occurred in 75 per cent of the cases, vomiting in only 8 per cent. Frank hemorrhage occurred in 10 per cent, perforation in 5 per cent. In the colored series 48 per cent had pain and 33 per cent had vomiting. The frequency of vomiting is accounted for by the associated gastritis.

Frank hemorrhage occurred in the negro series in 35 per cent. The large incidence of this symptom is due to the advanced type of case and the use of a heavy unrestricted diet. For the same reason, perforation occurred in 21 per cent. Some of these cases gave a history of having eaten a large meal of boiled cabbage just before their perforation.

Hemoglobin Values.—In the white series, these ranged from 55 to 99 per cent. In the colored series, a somewhat lower range was observed. A lower blood pressure range was also found in the colored cases.

Gastric Chemistry.—Hyperchlorhydria was preponderant in the whites, while hypo-acidity and achlorhydria occurred more often in the colored series.

Roentgen Findings.—The x-ray, of course, furnished the most conclusive findings in both series. In the diagnosis of gastric ulcer the most skilful x-ray interpretation is needed and a positive or negative diagnosis is significant. It is my belief that the single lesion occurs in far the greater number of cases of peptic ulcer in the Southeast, and that the multiple type of lesion is rare. While the duodenal lesion occurs as frequently in the Southeast as elsewhere, gastric ulcer occurs less frequently. Many cases, too, in the past, have been diagnosed gastric ulcer when the pathologic condition lay in the gallbladder or appendix.

Associated Diseases.—In the white series, one finds the spastic, irritable colon and a cholecystitis often associated with ulcer. Twenty-four cases had some history of appendical disease.

In the colored race it has been our observation that gallbladder disease and appendicitis are infrequent. Boland, who made a survey of appendical disease occurring in the City of Atlanta, states that thirty years ago appendicitis was almost a rarity in the negro, but that it is becoming more frequent because of his diet. There were only 3 cases of associated appendicitis in the colored series. Hookworm disease was found in three patients of the white ulcer cases. This disease may mimic peptic ulcer in that it sometimes gives rise to vague epigastric pains, due to the invasion of the duodenum by the parasites. A careful microscopic study of the stools should be made routinely in all chronic gastro-intestinal patients in the Southeast.

Cardiovascular Disease.—In the colored series, 13 cases of arteriosclerosis, cardiac decompensation and hypertensive disease were found associated with the ulcer cases. Recalling the large number of hemorrhages in the series, the contributory factor of this vascular complication presents an interesting conjecture. Cases of massive hematemesis in the arteriosclerotic individual have been reported in the literature.

Dietary History.—The dietary history of these two series of ulcer patients is entirely in keeping with the unbalanced food habits of this part of the country, which were mentioned earlier in this paper. In the white series, 33 gave a history of excessive use of cereals and sugars, 26 used no fruit or vegetables, 9 had an inadequate protein intake, and comparatively little milk was used by the majority. Many gave a history of too many soft drinks between meals. The dietary of the Southern negro is in the worst possible status. It is adequate only so far as it furnishes sufficient calories and satisfies his appetite. It comprises in its entirety all of the worst features of the white man's modern day diet, and is practically totally lacking in all protective factors. It universally consists of white flour bread and cornbread, fried pork and much lard, boiled cabbage and a great deal of syrup, sugar and other sweets. There are no fruits, raw vegetables, milk, butter and a minimum of fresh meat. From an economic standpoint they cannot afford these foods, and they can get cheaply white flour, rice, sugar, lard and pork. Here we have a race of people only three hundred years out of an aboriginal habitat, who follow their own inclination and appetites as far as eating is concerned, and who are subsisting on a food intake predominantly avitaminized, demineralized and of an acid-ash type, resulting in a lowered body resistance, metabolic disturbances and an increased susceptibility to chronic infections.

A frequent chronicity factor in the ulcer individual is a chronic colon stasis, it being present in 80 per cent of the white series and in 36 per cent of the colored. They have spastic colons, which apparently are a part and product of the vagotonic manifestations which occur in these people.

Complications.—In the white series, pyloric obstruction occurred in 8 per cent, and in the colored series in 8 per cent. Peritonitis occurred also in the latter cases in 2 per cent.

In the white series there was no association with syphilis, while in the colored series it was present in 29 per cent. This is about the average general incidence of syphilis among all admissions to the medical wards (colored), which is about 30 per cent. The actual incidence

of latent syphilis among these people is of course a great deal higher.

It has been pointed out that syphilis has no etiologic relation to peptic ulcer, and that peptic ulcer occurring in luetic individuals yields to treatment along the same lines as for a non-luetic individual.

Occupation.—In the white series the occupations were those found among the upper classes in any city. In the colored series, the occupations were those to be found in the general medical wards of any charity hospital and were made up of laborers, cooks, chauffeurs, maids, and so on. One observes here that heavy physical labor certainly exerts a very unfavorable influence upon the progress of peptic ulcer, as evidenced by the large number of hemorrhagic and perforated cases in the negro series.

Blood chemical analyses done on practically all of the 100 colored cases showed the blood sugar to be normal, and also the non-protein nitrogen and creatinine.

Management.—In the white series 80 per cent were treated medically and 10 per cent surgically. In the colored series, 65 per cent were treated medically and 30 per cent surgically.

Ultimate Outcome.—In the white series, there was improvement in all cases, both medical and surgical, with one death among the surgical cases. In the colored series, 68 per cent were improved and 10 per cent died.

CONCLUSIONS

One observes that in the general run of ambulatory ulcer patients in the Southeast gastric ulcer is rare and that duodenal ulcer is the predominant lesion; also that these lesions are single, and are comparatively free from such complications as hemorrhage in the large majority of cases. This may be due to the customary low residue diet used by these patients even before they come under observation. They yield very satisfactorily to the ambulatory type of management. On the other hand, the colored patients who made up the hospital cases in this study showed the greatest incidence of complications, because they were the severe and neglected types. They were hospitalized on account of complications such as hemorrhage, perforation, and high-grade pyloric obstruction.

Mann and Bollman point out: "The character of the food is also of significance in the development of the experimental ulcer. We have found that a diet of coarse food increases the incidence of ulcer in the animal with Eck fistula. Frauley and Ivy found that a coarse diet retarded the healing of the gastric mucosa of the rabbit from which a certain area had been denuded surgically. In our experiments perforation of an ulcer occurred at the time of passage from the stomach of rough objects, as noted by McCann."

Peptic ulcer obviously is a difficult problem in the negro race. In the first place the protective foods are economically out of his reach, neither do they come within his fancy from the standpoint of his appetite. Once free from his epigastric pain, he reverts to his erstwhile food habits. Usually also little is done to eliminate his oral sepsis. Therefore the chronicity factors go on. On account of these facts it seems to me that surgical measures in many cases offer the negro the best ultimate outcome, particularly for the prevention of hemorrhage and perforation.

One can offer the most to these patients by critical evaluation of the ulcer bearing individual himself, of his heredity, his psychology, and his environment and habits. And so while waiting for the actual etiological facts which yet remain unknown, one can remove and control the chronicity factors and help to restore the patient to the normal.

For too long, it seems, the mere mention of peptic ulcer has simultaneously envisioned the Sippy diet or a gastro-enterostomy, as if they were the alpha and omega of all therapy in this disease. As good and helpful as these two measures are, one cannot but feel that these patients are entitled to much more and to thoughtful consideration for a prolonged period of time.

DISCUSSION (*Abstract*)

Dr. C. W. Dowden, Louisville, Ky.—Our knowledge concerning the treatment of peptic ulcer has advanced but little in the past twenty-five years. The Sippy diet, without intention of detracting from that brilliant man's contributions, is, as we all know, the outgrowth of the Lenhartz system of diet. Complete alkalinization has been recognized for years as necessary for the relief of symptoms. Belladonna holds a place of doubtful value. It is now a proven fact that rest in bed with a complete change of environment will in many cases completely relieve the symptoms and in all cases prove a most helpful adjunct to any form of treatment. This would indicate that there is a constitution, congenital or acquired, that predisposes individuals to ulcer formation. I have an increasing conviction that too much attention is paid to the patient with an ulcer and not enough thought given to him as an individual before his ulcer has appeared.

TREND OF CASES OF SYPHILIS UNDER TREATMENT OR OBSERVATION IN THE UNITED STATES*

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Since the spring of 1927 the United States Public Health Service, in cooperation with state and city health authorities, has been making an effort to measure the effectiveness of present day syphilis control measures, to be expressed in the difference in the case rate per 1,000 population of those under treatment or observation for syphilis on a given census day as compared with the case rate for the same community after a three year interval. If there were no untreated, self-treated, or inadequately treated syphilis in the United States a downward trend of the prevalence rate could be translated immediately into terms of successful efforts in this field, but in the light of all these indeterminate factors frequently the upward trend of the curve is paradoxically indicative of the most fruitful results. Data on the extent of the problem in typical rural and urban communities throughout the United States have been published in "Venereal Disease Information" during the past few years and are used in the present study to form a base line from which to determine the trend during the three years interim, 1927-1930,[1] which has been calculated from the number of cases of syphilis in 16 communities actively under treatment or observation in 1927 as compared with 1930. In 14 of the communities comprising 8 cities ranging in size from 16,400 in El Dorado, Arkansas, to 105,000 in Peoria, Illinois, and in six counties in West Virginia, Kentucky and Illinois, with populations from 14,400 in Scott County, Kentucky, to 63,250 in Pike County, Kentucky, the combined changes in case rates of syphilis per 1,000 population showed a 4.4 per cent decrease. However, this downward trend is not a consistent one throughout each of the 14 communities, although it did occur in 9 of them.

In the other two communities in which statewide surveys were made, one in Oregon[2] and the other in up-state New York,[3] there was a definite increase in the syphilis rate, in Oregon 18 per cent and in up-state New York 15 per cent. It is interesting to note that in the total rate for 14 communities the decrease was among the late or chronic cases under treatment and not among the early cases. As a matter of fact the composite change in rates in the 14 communities revealed a 13 per cent increase in the cases admitted to treatment while in the early stage of the disease.

A similar situation was found in Oregon for although there was an increase in case rates per 1,000 population among both the early and late cases of syphilis under treatment or observation, the percentage increase among the early cases was 38 per cent as compared with 10 per cent increase in the late.

Probably the most encouraging findings in the resurveys are those rather appreciable increases in the number of early cases of syphilis which come under treatment. Certainly nothing will ultimately so surely bring under control this elusive disease as will the early recognition and treatment by both the private practitioner and the public clinic. Many factors may be responsible for the bringing of a larger group of cases under treatment while the infection is in the early stages. Among these may be mentioned the provision of more adequate public clinic facilities for the part pay as well as indigent patient, a wider use of the state laboratories for performing the diagnostic tests, the influence of educational measures in the hands of the private physician and the public clinics, and the routine Wassermann testing of hospitalized persons.

It may be expected that the rate for chronic cases will gradually show a reduction as resurveys are continued at various intervals, first because of the growing tendency, in private practice, to make a routine Wassermann on all pregnant women, which should lead to the elimination of much of the congenital syphilis which makes up part of the chronic group today, and second, as early cases come in promptly and receive adequate treatment they should never reach the chronic stage, but effect a satisfactory permanent reversal of the blood Wassermann and destruction of the organism before it is deeply seeded in the various organs with the danger of late central nervous system, cardiovascular or visceral involvement. It is indeed

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encouraging to find in 14 communities that the percentage of cases coming to public clinics while the disease is in the early stage is 11 per cent higher than it was three years ago. The benefits of this attitude towards early treatment are incalculable.

METHOD OF RESURVEY

A complete discussion of the manner of carrying out the prevalence surveys was given in an article setting forth the prevalence of venereal disease in the United States, published in "Venereal Disease Information."[1] As nearly as possible the same procedure was carried out in the resurvey. Each person and institution in the resurveyed community authorized to treat the sick was supplied with a questionnaire on which to record the number of cases of syphilis under treatment or observation as of a given date. The requested data were classified by sex and color of the patient and stage of the disease. Syphilis was designated as "early" in all cases in which one year or less had elapsed since the probable date of infection, and as "late" in all cases where more than one year had elapsed. As far as was possible, all factors affecting the completeness and accuracy of the data were accounted for. The good faith and willingness to cooperate on the part of those who filled out the questionnaire was secured by seeking the approval of members of the medical profession through local health and medical associations. A personal representative of the Public Health Service did follow-up work for all missing questionnaires in fifteen of the communities.

Undoubtedly there are uncontrollable factors which do affect the data, but on the whole these contribute to understatement rather than overstatement of the problem. It is felt that the number of cases of syphilis reported as under treatment or observation represents the very minimum in the United States as well as showing with fair exactness the extent of syphilis as a causative factor in the population under medical care each day. The uncontrollable factors which would make comparison between different communities inadvisable are the inclusion of nonresident patients, the availability of free treatment, and the extensiveness and effectiveness of follow-up and social service in keeping the patient under care until the maximum benefits have been received, and the varying personal equation of physician and patient.

SIGNIFICANCE OF THE INDICATED TRENDS BASED ON REPORTS OF TREATMENT SOURCES OF A 24,500,000 POPULATION

The number of persons constantly under treatment or observation for syphilis in continental United States in 1927 was estimated at 643,000 cases.[1] This estimate was based on the cases reported as actually under treatment or observation for syphilis by all medical sources in 25 representative communities throughout the United States, which included 29,918 physicians, 828 other private practitioners, and 1,101 public institutions responsible for the medical care and treatment of 24,498,000 people. It is felt that the findings among 20 per cent of the nation's population selected from the most rural territory to the great metropolises of the country are a reliable index of the syphilitic population constantly under medical care day by day.

Resurvey data in 1930 for 16 of these communities with a population of approximately 7,000,000, including up-state New York, the State of Oregon, 8 cities of 100,000 or less population and 6 counties in four other states, Arkansas, Illinois, Kentucky, West Virginia, form the basis of this estimated trend. The trend for cases of syphilis under treatment or observation throughout the United States can be expressed as ranging from a possible decrease of 28,000 cases to a possible increase of 100,000. The difference in the trend in the various communities in 1927 as compared with 1930 shows that the individuals infected are still neglecting treatment rather than any definite increase or decrease in the actual number of persons being infected. These evidences of apparent indifference of the infected individual toward the treatment of syphilis are viewed with deep concern. While the population of some communities may be responding to the concentrated efforts of control officers to seek early and adequate treatment for syphilis, the great difference in the rates throughout this resurveyed territory reveals that it is not a universal response. There is much evidence that only a small part of the syphilitic population receives adequate treatment, or, as a matter of fact ever seeks treatment. Indeed, approximately 50 per cent of the syphilitics never seek any treatment until in the late stages even though in many instances public clinic facilities are available, as will be seen later in the discussion of the incidence of syphilis among a population of approximately 11,500,000 persons.

The difficulty of keeping syphilitics under treatment is well known and is experienced not only in private practice but in public clinics. Recent studies by the U.S. Public Health Service in eight of the best equipped syphilis clinics in the country revealed that two-thirds of the patients lapsed from treatment before the maximum benefits had been received[4 5] even though the disastrous consequences of inadequately treated syphilis are constantly stressed. It is quite well known that syphilis contributes 11 per cent of the first admissions to the institutions for mental diseases,[6] is an important causative factor in disabling heart conditions,[7] and is responsible for a high percentage of fetal deaths.[8] In fact, an average of 6.9 per cent of the pregnant women admitted to the maternity hospitals have a positive Wassermann.[9] Jeans reports that 24 per cent of the 3,009 pregnancies among 870 syphilitic families resulted in the death of the fetus on or before term.[10] Further evidence of the projected effects of syphilis can be seen in a study of 33,000 unselected rural negroes of the South who under routine Wassermann test revealed 25 congenital syphilitics per 1,000 population.[11]

A review of the clinic cases from 1927-1930 in up-state New York shows that the increase in number of cases of syphilis actually under treatment or observation at the end of the year is due to cases remaining under treatment for a longer period of time, thus receiving more adequate treatment. This conclusion was arrived at from examination of the number of new cases admitted during the three year period. The new clinic cases increased 5.7 per cent whereas the total number of cases at the end of the third year had increased about 59 per cent. Since there was a noticeable decrease in the number of early cases prematurely discontinuing treatment, it is concluded that at least for clinic cases in up-state New York prolongation of treatment, and not new admissions, is largely responsible for the increased number of syphilis patients constantly under clinic care.[3] Similar data in up-state New York were not available for physician's cases, but this situation in the clinics in certain localities leads to the belief that persons infected with syphilis are gradually acquiring a higher appreciation of the necessity of thorough treatment.

PREVALENCE RATES FOR SYPHILIS IN EACH OF 16 COMMUNITIES

During the three-year period there was in the total resurveyed territory a 6 per cent increase in the case rate per 1,000 population for patients seeking treatment within the first year of infection, and a corresponding increase of 14 per cent in the case rate for those coming to treatment in the late stage of the disease. Unfortunately this increase in case rates for those reporting for treatment in the early stage is not uniform throughout the 16 communities. In fact, in some communities the reverse is true. For example, in up-state New York there was a high rate of increase (23 per cent) among the late cases while among the early cases there was a 3 per cent decrease. In the State of Oregon, although the case rates for both early and late cases increased, the increase among the early cases was four times that of the late. The early cases in 5 of the 8 cities of 100,000 or less population showed a decrease in the case rate as also in two of the 6 counties included in the 14 communities resurveyed. However, the total case rates for the 14 communities had a 13 per cent increase among early cases and a 13 per cent decrease among the case rates of infected individuals reporting for treatment after the infection reached the late stage. The syphilis case rates per 1,000 population for the two cities of more than 75,000 population, Peoria, Illinois, and Huntington, West Virginia, both with good public clinic facilities, gives a very similar picture for the early cases of syphilis, both showing a high percentage of increase per 1,000 population. The total syphilis rate for Wheeling, West Virginia, and Decatur, Illinois, cities of 50,000 to 75,000 population, are 6.2 and 6.8 per 1,000 in 1930 as compared with a rate of 8.0 and 8.3 in 1927. This decrease in both cities occurred among the late cases. In the four cities with populations of 15,000 to 50,000, Lexington, Kentucky; El Dorado, Arkansas; Paducah, Kentucky; and Texarkana, Arkansas, the syphilis rates are two and one-half to five times higher for Lexington than they are for the other three cities and yet Lexington is the only one which maintains a public clinic. This high rate is undoubtedly not due to an excessive colored population as all four of these cities have approximately the same high percentage of colored population.

Does the knowledge that free treatment for syphilis is available make the population any less fearful of the consequences of the disease? Is it possible that greater liberty and license exists in one town than in another of approximately the same size? The results of recent studies to a large extent discount the importance

of these causes in the increase of syphilis, which revealed that only one in 25 negroes with a positive blood Wassermann seeks treatment for his syphilis,[11] and that not more than 50 per cent of the white clinic population ever seek treatment for syphilis until after it reaches the late stages. With all these untreated syphilitics in any community it is believed any fresh or renewed effort, either in the field of education or medicine, should result in bringing to treatment many of those who have been inadequately treated or who remain entirely untreated for syphilis. With the present knowledge of the importance of rendering a fresh case of syphilis free from the possibilities of infectious relapse, the desirability of early and adequate treatment for syphilis cannot be too strongly emphasized.[12] Further, an increase in the number of cases of syphilis under treatment at any one time cannot be regarded with anything but the greatest optimism. The above referred to studies, disclosing the tremendous number of untreated syphilitics, were made in quite rural communities, which fact invalidates much of the popular belief that syphilis is less prevalent in rural than in urban areas. In the 14 communities under consideration the prevalence rate for syphilis per 1,000 population in the rural area was slightly higher in 1930 than in 1927. It is still less than one-half as high as the urban rate. Further, the counties which have public clinic facilities have a higher prevalence rate. Thus it would appear that there is much to be done in training the rural physicians and populations in the recognition, treatment, and necessity for the treatment of early syphilis.

EFFECT OF SEX ON CASE RATES FOR SYPHILIS PER 1,000 POPULATION

The case rates per 1,000 population under treatment for syphilis are consistently higher for males than for females in the resurveyed territory. In the total resurveyed territory the case rate per 1,000 population for males is 3.8 as compared with the case rate for females of 2.3 per 1,000 female population. It is interesting to note that although the case rate for females increased 26 per cent more than did the rate for males in the three year period, it is still a third less than the present rate for males. These rates do not necessarily mean that there is less syphilis among females; they do show that it is being recognized, probably the result of routine Wassermann tests. Among both sexes there are more patients seeking treatment after their infection has reached the chronic stage than come in while the disease is in the early stages. In fifteen of the communities where the data are available the tendency in the past three years has been to report earlier for treatment. Considerable improvement among the males reporting for treatment early was seen in Hunt-

Table 1

INFLUENCE OF STAGE OF INFECTION ON ADMISSION ON CASE RATES PER 1,000 POPULATION FOR SYPHILIS SHOWING PERCENTAGE OF INCREASE OR DECREASE, 1930 AS COMPARED WITH 1927

	Early			Late			Total		
	1927	1930	Per Cent Increase or Decrease	1927	1930	Per Cent Increase or Decrease	1927	1930	Per Cent Increase or Decrease
14 communities Number	1423	1751	23.05	2692	2535	−5.83	4115	4286	4.15
Case rate	2.36	2.67	13.14	4.47	3.87	−13.42	6.83	6.54	−4.25
Oregon Number	567	857	51.15	1527	1821	19.25	2094	2678	27.89
Case rate	.65	.90	38.46	1.74	1.91	9.77	2.39	2.81	17.57
Up-State New York Number	3713	3791	2.07	9223	11941	29.47	12936	15732	21.61
Case rate	.68	.66	−2.94	1.68	2.06	22.62	2.36	2.72	15.25
Total Number	5703	6399	12.20	13442	16297	21.24	19145	22696	18.55
Case rate	.82	.87	6.10	1.93	2.20	13.99	2.75	3.07	11.64

Table 2

EFFECT OF SEX ON CASE RATES PER 1,000 POPULATION FOR SYPHILIS, SHOWING PERCENTAGE OF INCREASE OR DECREASE, 1930 AS COMPARED WITH 1927

	Males		Per Cent Increase or Decrease	Females		Per Cent Increase or Decrease	Total		Per Cent Increase or Decrease
	1927	1930		1927	1930		1927	1930	
14 communities Number	2524	2672	5.86	1591	1614	1.45	4115	4286	4.16
Case rate	8.31	8.18	−1.56	5.34	4.91	−8.05	6.84	6.54	−4.39
Oregon Number	1366	1744	27.67	728	934	28.30	2094	2678	27.89
Case rate	2.93	3.49	19.11	1.77	2.06	16.38	2.39	2.81	17.57
Up-State New York Number	8091	9588	18.50	4845	6144	26.81	12936	15732	21.61
Case rate	2.97	3.35	12.79	1.75	2.10	20.00	2.36	2.72	15.25
Total Number	11981	14004	7.88	7164	8692	21.33	19145	22696	18.55
Case rate	3.43	3.80	10.79	2.06	2.34	13.59	2.75	3.07	11.64

ington, West Virginia; Texarkana, Arkansas; Logan County, West Virginia; and Scott County, Kentucky. In the 1927 survey a higher percentage of the males came to treatment for syphilis after the first year of the infection, whereas in the 1930 survey the percentage of males seeking treatment in the first year of the disease in the above mentioned communities was nearly twice the number who sought treatment after the infection had reached the late stages. This condition cannot be taken to mean that the number of infections is decreasing for in two of these four communities (Texarkana, Ark., and Scott County, Ky.) there was an actual decrease in the total syphilis rate for the males, but it does mean that at least the patient is beginning to recognize the advantages of early treatment.

Table 3

THE PERCENTAGE OF CASE RATES PER 1,000 POPULATION FOR SYPHILIS TREATED IN PUBLIC CLINICS AND PRIVATE PRACTICE, BY STAGE OF INFECTION IN 1930

Community	Public Clinics			Private Practice		
	Total	Early	Late	Total	Early	Late
8 cities, 100,000 or less population Case rates	4.27	1.36	2.91	4.15	1.93	2.22
Per cent	50.7	31.9	68.1	49.3	46.5	53.5
6 counties Case rates	.91	.37	.54	2.22	1.19	1.03
Per Cent	29.1	40.7	59.3	70.9	53.6	46.4
Oregon Case rates	.47	.12	.35	2.34	.78	1.56
Per Cent	16.7	25.5	74.5	83.3	33.3	66.7
Up-State New York Case rates	1.04	.21	.83	1.68	.45	1.23
Per Cent	38.2	20.2	79.8	61.8	26.8	73.2
Total Case rates	1.15	.27	.88	1.92	.60	1.32
Per cent	37.5	23.5	76.5	62.5	31.3	68.7

DISTRIBUTION OF CASES IN PUBLIC CLINICS AS COMPARED WITH PRIVATE PRACTICE

In 1930 among these 16 resurveyed communities, 62.5 per cent of the patients were in the hands of private practitioners as compared with 37.5 per cent under treatment in public clinics. During the three year period there was a shifting of 7 per cent of the cases from private practice to public clinics in the total surveyed territory. (The most significant transition of private practice cases to public clinics was in Huntington, West Virginia, Paducah, and Pike County, Kentucky, the change representing about 33 per cent of the cases.) Undoubtedly the present economic depression is in a large measure responsible for the increased number of syphilitics seeking treatment in public clinics. In 14 of the communities data by stage of infection are available for the public clinic and private practice cases. In the six counties the increase was 18 per cent among the cases com-

ing to the clinics early for treatment but the proportion of cases seeking treatment early decreased in the cities of 100,000 or less population.

In the total resurveyed territory the early cases of syphilis comprised 23.5 per cent of the cases of syphilis under treatment in public clinics and 31.3 per cent of those in private practice. Obviously too many infected individuals are not seeking treatment until after their disease is in the late stages.

ESTIMATED ANNUAL INCIDENCE OF FRESH INFECTIONS OF SYPHILIS

In the more recent prevalence studies there has been included in the questionnaire a request for the number of patients with syphilis who reported for the first time for observation or treatment during the preceding month. These data were assembled as the basis for estimating the number of fresh infections occurring annually as well as to determine the percentage of syphilitics who sought treatment during the early stage of the infection. The month preceding the prevalence survey date was selected for recording the incidence data because of its convenience to those filling out the questionnaire and also because it is felt that one month is as representative as another in the absence of any seasonal variation in the disease.

Philadelphia was the first community sur-

Table 4

ANNUAL INCIDENCE RATES PER 1,000 POPULATION FOR SYPHILIS IN 1930 AMONG A POPULATION OF 11,740,561 PERSONS

Locality	Number of Cases			Rates per 1,000			
	Total	Early	Late	Total	Early	Late	Population
Oregon State	4,056	1,848	2,208	4.25	1.94	2.31	953,786
Up-State New York	25,524	10,752	14,772	4.41	1.86	2.55	5,787,805
Philadelphia, Pa.	19,344	7,980	11,364	9.37	3.87	5.50	2,064,200
Charleston, W. Va.*	2,508	1,440	1,068	41.52	23.84	17.68	60,411
New Orleans, La.	6,528	3,744	2,784	14.23	8.16	6.07	458,762
Dallas, Tex.	7,524	4,836	2,688	28.89	18.57	10.32	260,475
Birmingham, Ala.	7,344	2,556	4,788	28.28	9.84	18.44	259,678
Jefferson County, Ala. (exclusive of Birmingham)	1,872	996	876	10.90	5.80	5.10	171,815
Baltimore, Md.	8,460	3,972	4,488	10.51	4.93	5.58	804,874
Five counties in Maryland	1,224	636	588	4.64	2.41	2.23	263,482
14 communities—total	8,604	6,036	2,568	13.13	9.21	3.92	655,273
14 communities in detail							
Peoria, Ill.	1,320	840	480	12.57	8.00	4.57	104,969
Huntington, W. Va.	1,164	936	228	15.40	12.38	3.02	75,572
Wheeling, W. Va.	648	312	336	10.51	5.06	5.45	61,659
Decatur, Ill.	216	156	60	3.75	2.71	1.04	57,510
Lexington, Ky.	1,572	1,236	336	34.37	27.02	7.35	45,736
Eldorado, Ark.	468	216	252	28.50	13.15	15.35	16,421
Paducah, Ky.	276	120	156	8.23	3.58	4.65	33,541
Texarkana, Ark.	468	360	108	17.10	13.15	3.95	27,366
LaSalle District, Ill.	60	60	.....	2.29	2.29	.....	26,180
Pike County, Ky.	504	432	72	7.97	6.83	1.14	63,267
Logan County, W. Va.	1,596	1,140	456	27.27	19.48	7.79	58,534
Morgan County, Ill.	12	.....	12	.35	.....	.35	34,240
Greenbrier County, W. Va.	72	24	48	2.01	.67	1.34	35,878
Scott County, Ky.	228	204	24	15.83	14.17	1.66	14,400
Total	92,988	44,796	48,192	7.92	3.82	4.10	11,740,561

*Exclusive of Charleston City Clinic cases for which data by stage of infection were not available.

veyed which had included in the questionnaire an inquiry into the incidence of the disease. Similar data have now been collected from 24 communities with a population of 11,740,561, or 9.4 per cent of the total population of continental United States. The urban population is proportionately higher than is desirable to be comparable with the division of the data into urban and rural for the country as a whole. However, the data here presented represent a fairly good distribution between rural and urban population, as well as geographical representation. In Oregon State, 49 per cent of the population surveyed is rural while in up-state New York there is about an equal division between rural and urban territory, ranging in population from small towns to cities of a half million. Most of the communities in the 14 studied have a high percentage of negroes which explains in a large degree the inflation in the case rates in these communities. In the original estimate based on the findings in Philadelphia alone, the information was confined to the rate per 1,000 persons reporting with early infections. It was felt that in this way at least a minimum of fresh infections occurring annually would be shown. It is indeed interesting that with more than five times the population resurveyed, including persons from rural areas as well as a large percentage of colored population, the composite rate for the incidence of fresh infections of syphilis is practically unchanged. These rates for early syphilis ranging from less than 1 per 1,000 population to 24 per 1,000 population give a resultant rate which very closely approximates that found in Philadelphia and thus gives added weight to the estimate of 423,000 cases of fresh infections each year based on the Philadelphia incidence data. It is also of interest that for every case of syphilis reporting for treatment in the early stages of the disease one case delays seeking treatment until the disease has reached at least the second year of infection. The seriousness of this neglect is reflected not only in the lessening of chances for cure for the infected individual but also in the greater danger to the community from the untreated resident syphilitics.

SUMMARY

A downward slope cannot be translated into successful efforts in the field of control of syphilis because of the large number of untreated, self-treated, and inadequately treated cases of syphilis which any renewed effort in the field of education or medicine in a community brings under treatment. The great variation in the changes in prevalence rates in the three year interim (1927-1930) in the 16 communities resurveyed emphasizes the validity of this statement.

The changed rate among the 16 communities comprising 7,000,000 population ranges from a downward trend of 4.4 per cent in the composite 14 smaller communities to an upward trend in two state wide resurveys of 15 to 18 per cent.

In 15 of the communities there was a definite increase in the early cases coming to treatment, which fact is probably the most encouraging finding in the resurvey. Regardless of how many additional fresh infections occur, the disease will eventually be brought under control if the populace can be educated to come early for treatment of syphilis and remain until the disease is rendered noninfectious. There is still much to be done in this direction for although there is an increase in the early cases coming to treatment there are still approximately one-half of the infected individuals who never seek treatment until the disease is in the late stages.

Based on the 1927 prevalence surveys of syphilis under the care of medical sources treating approximately 24,500,000 of the nation's population, it was estimated that there were 643,000 cases of syphilis constantly under treatment. Three years later a resurvey of the medical sources treating approximately 7,000,000 of the same population gave a wide difference in rates in the several communities. Based on the total findings of these prevalence surveys it is estimated that the syphilitic population constantly under medical care day by day changed from a possible decrease of 28,000 cases in the 14 rural communities, to a possible increase of 100,000 cases in up-state New York and Oregon in the three year period.

Some of the reasons for these variations have been attributed to the successful efforts of the health officers in getting more of the untreated or self-treated syphilitic population under treatment. In up-state New York the investigator claims that the increase is due to successful efforts in keeping syphilis patients under treatment for a prolonged period of time until the maximum benefits of treatment have been received, claiming that thus the increased patient population in clinics is due to an accumulation of cases.

An upward trend of the prevalence curve with either of these causes the determining factor would be most encouraging.

In the total resurveyed territory there was a 6 per cent increase in the case rates per 1,000 population among the early cases and a 14 per cent increase among the late cases.

The availability of free clinic facilities appears to carry with it higher prevalence rates.

The rural rates are not as high as the urban in the 14 communities surveyed.

The case rates for males are consistently higher throughout all communities than they are for females. It is interesting that although the case rate for females increased 26 per cent more than did the rate for males in the three year period, it is still one-third less than the present rate for males.

In 1930 in the 16 communities resurveyed, 62.5 per cent of the patients were in the hands of private physicians as compared with 37.5 per cent under treatment in public clinics. During the three year period there was a shifting of 7 per cent of the private practice cases to public clinics, probably due in large measure to the present economic depression.

The early cases of syphilis were 23.5 per cent of the cases of syphilis under treatment in public clinics and 31.3 per cent of those in private practice.

Data on the number of fresh infections reporting annually for treatment have been gathered from medical sources treating 9.4 per cent of the nation's population. The estimate based on Philadelphia alone made in 1929 is supported by the composite incidence rates in these 24 communities. Thus the estimated incidence of fresh cases reporting for treatment each year in continental United States is 423,000 cases. However, there is an equally large number who are also reporting for treatment each year for first time treatment of a late syphilitic infection.

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DISCUSSION (*Abstract*)

Dr. Hugh J. Morgan, Nashville, Tenn.—The work that has been carried on by Drs. Parran, Clark, Usilton and associates in the United States Public Health Service is of the greatest importance. As a result of their data we are able to arrive at a fairly accurate estimate of the volume of syphilis in the United States. Before their reports became available there was no reliable information on the subject. This paper is but one of a series of extremely important communications.

Dr. D. C. Smith, University, Va.—The increased number of cases of syphilis under treatment in this country during the last several years may cause one to feel pessimistic about the disease. The increased number of cases under treatment is, however, thought due to an improvement in the discovery of cases with syphilis.

The efforts in regard to the handling of syphilis in this country since the World War have been just about sufficient to care for the average number of new cases. As the proportion of preventive measures increases beyond the point at which the above situation occurs the trend of syphilis will be downward. Whenever the point is reached where the decrease begins to occur then a very slight increase in the effort of eradication will accelerate the decrease in the disease.

OCULAR NEUROPATHIES AND AMAUROSES IN MENINGOCOCCIC MENINGITIS*

By PHIL. M. LEWIS, M.D.,
Memphis, Tenn.

During the past few years a large number of cases of epidemic cerebrospinal meningitis have occurred in and around the City of Memphis. The majority of these cases were treated in the Isolation Department of the Memphis General Hospital. A report[1] on the eye findings in the early part of the epidemic was read before this Section in 1930. Since May 1, 1930, I have examined the eyes of all cases except some of those who died within a few hours of admission. The total number of these patients whose eyes I have studied amounts to 184. A few

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*From the Department of Ophthalmology, University of Tennessee.

cases have shown changes in the optic nerves and visual pathways which I believe are sufficiently interesting to call to your attention.

FREQUENCY AND TYPE OF LESIONS

It is surprising, considering the proximity of the disease process, that involvement of the optic and other cranial nerves does not occur much more frequently than it actually does in cerebrospinal meningitis. Randolph[2] reported optic neuritis in 6 out of 40 cases, or 15 per cent. Uhthof[3] found optic neuritis in 18 cases out of 110, or about the same per cent as Randolph. Wissmann[4] observed 7 cases of actual optic neuritis, several of hyperemia and one case with an homonymous hemianopia, associated with facial paralysis and motor and sensory disturbances on the left side of the body. In our series we had only 7 cases, or 3.8 per cent, of definite neuritis. However, we had 21 cases of hyperemia of the discs and retina. Some of these might easily have been classified as neuritis, although no elevation of the discs was found. If classed as neuritis they would bring our total to 28 cases, or 15 per cent, which would make our figures more in line with other observers.

There were 3 patients, or 1.6 per cent, of the total number who had complete amaurosis. Two of these were infants in whom it is difficult to prove absolute blindness. The third was an adult reported in full below.

One infant showed considerable pallor of both nerve heads, with a hazy, irregular or worm-eaten appearance of the margins such as is found in postneuritic optic atrophy. This obviously could not be attributed to meningitis, as the child had been ill only a few days. Optic atrophy was not seen in any other patients while in the hospital, but follow-up examinations two or more months later revealed it in varying degrees in every case of optic neuritis which could be traced and in the one living patient who had a complete temporary amaurosis.

In 17 patients the fundi could not be examined due to cloudiness of the media from endophthalmitis, keratitis and other conditions.

Table 1

OPTIC NERVE FINDINGS IN 184 CASES OF EPIDEMIC CEREBROSPINAL MENINGITIS

Diagnosis	No. Cases	Percentage
Normal eye findings	89	43
Fundi not visible	17	9
Hyperemia of disc and retina	21	11.2
Optic neuritis	7	3.8
Amaurosis	3	1.6
Early optic atrophy	0	0
Late optic atrophy	4*	Undetermined

*Unable to follow other cases.

OPTIC NEURITIS

From our series of cases one must conclude that real optic neuritis is certainly not very frequent in meningococcic meningitis. Only 7 in 184 casts, or 3.8 per cent, is considerably less than the findings of other observers. As was pointed out previously, however, 21 patients showed a definite hyperemia of the discs. The discs showed no elevation in these cases, but they were abnormally red and might by some observers have been classified as true neuritis. The line drawn between the two is undoubtedly very fine in some cases.

The elevation of the discs was not great in our series, usually from ½ to 1½ diopters. This is in accord with the findings of most observers. One patient who died had 3 to 4 diopters of swelling and numerous hemorrhages. Autopsy showed a massive fibrinopurulent exudate covering the entire base of the brain, most pronounced in the region of the chiasm and around the cerebellum. The ventricles were all distended and the canal of the spinal cord was closed with purulent exudate. Increased intracranial pressure was evidently responsible for the marked swelling of the papillae in this case.

The color of the discs was not characteristic, the degree of redness being similar to that ordinarily seen in neuritis. In several cases small hemorrhages occurred near the border of the discs and extended out into the retina, following the nerve fibers. All of the cases in our series were bilateral, though as a rule on one side the process was more pronounced than on the other. Of the 6 cases with neuritis which lived, all recovered fairly good, useful vision. A partial secondary optic atrophy was noted in 3 cases which I had the opportunity of following. Sector-like defects in the fields of one or both eyes were not unusual. The prognosis regarding useful vision, however, is good.

A report of a typical severe case of optic neuritis follows:

H. R., a white man, age 29 years, was admitted to the Isolation Hospital on February 1, 1931, with meningococcic meningitis. The usual physical and laboratory findings of acute epidemic cerebrospinal meningitis were present and the customary treatment was started. Two days later I made the routine ophthalmoscopic examination, the pupils being dilated with homatropine. I found the retinal vessels tortuous, the veins dilated and the papillae quite red but not elevated. I recorded on his chart that he had a simple hyperemia of the discs and retinae. On February 8, 1931, the condition had passed beyond the stage of simple hyperemia. Both discs were slightly elevated and the margins were indistinct. Two days later, when he was next examined, the neuritis was increasing. On February 13,

1931, the elevation of the right disc was about ¾ diopter and that of the left about 1½ diopters. The margins were greatly blurred and a few small hemorrhages were noted on the papillae. The neuritis remained about the same until February 19, 1931, when it began to subside slowly. On this date, the twenty-second after his illness began, he was improved sufficiently to take his vision. It was found to be reduced to hand movements in both eyes. Six days later the fundi were greatly improved, the elevation of the discs had almost disappeared, but vision was still only hand movements. Improvement in vision was soon noted, however, and by March 10, six weeks after his illness began, he could see to read large print. The appearance of his fundi was at this time practically normal.

The patient was dismissed from the hospital March 14. He was very shaky on his feet, but could walk around slowly. He reported for examination of his eyes as soon as he was physically able, which was on April 4, 1931. At that time vision had improved to 20/60 in the right eye and 20/30 in the left. The margins of the papillae were indistinct, otherwise the eyes appeared completely normal. Almost the entire lower field of the right eye was lost and there was a moderate contraction of the temporal field (*Fig. 1*).

The field of the left eye was normal; there were no scotomata and the blind spot was normal. On April 27, over ten weeks after the neuritis was at its summit, the signs of optic atrophy first made their appearance and then only in the right eye. The margins of both discs were irregular and a little indistinct, but only the right eye showed a definite pallor. On this date the fields were found to be about the same. Vision had not improved. Accommodation was weak, being only 5 diopters. One month later improvement was noted. Vision was 20/50 in the right eye and 20/20—3 in the left. The field of the right eye was improved (*Fig. 2*), but a considerable defect in the lower half of the field was still present. This defect has remained the same and is apparently permanent, as is the diminished vision of the right eye and the lowered accommodation of both eyes.

When last seen, ten months after his illness, both discs appeared rather white throughout, although the vision and other subjective findings were unchanged and will evidently so remain. He is able to work regularly at his occupation of starter for a bus company.

Treatment. — This man was so ill that death was expected daily. The note on his chart by the house officer in charge said, "Prognosis hopeless," day after day. He received 18 intraspinal injections of antimeningococcic serum, a total of 480 c. c. He was given 60 c. c. intravenously, as well as glucose solution intravenously and one 500 c. c. whole blood transfusion. In spite of his desperate condition his temperature never exceeded 101° F. by axilla. Often it was as low as 96° F. and for several days in succession it did not exceed 97° F. This picture is not unusual in this disease. High temperatures are the exception rather than the rule.

The duty of the ophthalmologist in these cases is to examine the fundi every few days or even daily in severe cases, noting any changes that occur, especially an increasing elevation of the discs. In this way the attending physician may have an additional guide as to the frequency of doing lumbar or cisterna punctures. Very severe or complete optic atrophy may in certain cases be thereby prevented.

AMAUROSES

Three cases with complete blindness but normal ophthalmoscopic findings occurred in our series. Two were infants, both of whom died, one after 43 days in the hospital and the other after 25 days. Autopsy was refused in the first and did not reveal the cause of the blindness in the latter. Distended ventricles and numerous adhesions along the base of the brain were found, but unfortunately sufficient investigation of the optic pathways was not made.

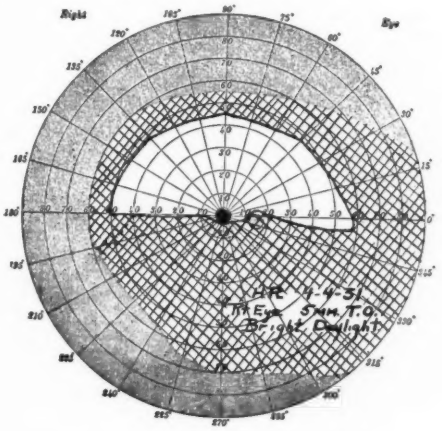

Fig. 1
Visual field of Case H. R. on April 4, 1931

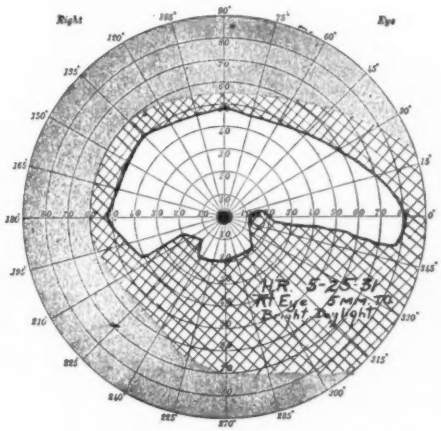

Fig. 2
Visual field of Case H. R. on May 25, 1931

The case which recovered has been quite interesting and is therefore reported.

W. N., a colored man, age 21 years, was admitted to the Isolation Hospital on April 17, 1930, with meningococcic meningitis. The usual physical and bacteriological findings of the disease were present and the customary treatment started. Now at that time I had not begun to examine the eyes of all these cases routinely, so did not see this man until he became blind. Previous to his blindness he had run an usual sort of course, being clear mentally at times, frequently semicomatose and at times highly delirious. On the morning of the sixteenth day of his illness he awoke to find he could not see at all. Prior to this he had noticed nothing wrong with his eyes and the examination by the house officer on admission had shown his eyes normal. I examined his eyes on the day following the onset of blindness and found he could not perceive even a very bright light in either eye. The pupils were dilated and did not react. Otherwise his eyes were practically normal, there being no cloudiness of the

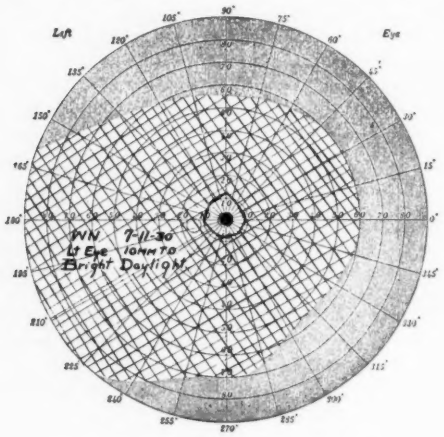

Fig. 3
Visual field of Case W. N. on July 11, 1930

media and nothing abnormal in the fundi except a scleral ring or small inferior conus of both discs. No orbital pain or tenderness was present. Complete blindness with normal fundi remained for almost three weeks and then light perception returned. Except for the blindness he recovered perfectly from the meningitis and was discharged from the hospital on May 23, 1930, a litt'e over five weeks after admission. At that time he had light perception in the right eye only in the superior field, while in the left eye he could perceive light in all fields except the nasal. The media and fundi were still normal.

On June 6, 1930, he was seen in the outpatient department. Vision was the same in the right eye, but improved in the left to counting fingers at 3 feet. Three weeks later the right eye could see hand movements straight in front of it, and the left eye 20/200. The fundi were still normal when next seen on July 11. The right eye had improved to counting fingers at 3 feet and the left to 20/30. It was noticed that the patient had difficulty in getting around and on testing the fields they were found to be practically telescopic. Colors were recognized only centrally. Vision of the right eye was so poor that recording of the field was impractical. By confrontation, however, there was no vision except a small central area.

The patient was next seen on August 1, and a pallor of the right disc was present. This was the first manifestation of any pathological condition in either fundus. The left disc appeared normal. Vision and fields were practically unchanged. On September 11, definite atrophy of the right disc was present and the left was thought to show a beginning pallor. The atrophy of the right disc was of the primary variety. Vision had improved very slightly in both eyes. The field of the right eye was only a small central area, less than five degrees in size. The left was unchanged. The patient was not seen again until January 9, 1931. Vision was then in the right eye 20/50, in the left 20/30 plus. The right field was still telescopic, the left had improved considerably. With a very large test object (20 mm.) the field limits of the left eye were only about ten degrees below normal except in the lower nasal quadrant, where he was completely blind. Both discs were now white, moderately cupped and the lamina cribrosa plainly visible. The blood vessels showed no appreciable change.

The patient was not seen again until May 5, 1931, just one year after his illness. His vision with correct lenses (+.50 cyl. X 180 O. U.) was 20/40 in the right eye and 20/20—2 in the left. The fields had improved, but the right was still very poor and the left still had an absolute scotoma of the entire lower nasal quadrant. No color was perceived at all with the right eye except red and that only centrally. With the left eye color vision was good centrally, but was lost beyond 8 degrees. Fields taken several times since May, 1931, have shown no appreciable difference, so evidently no further changes will occur. The discs also have remained the same.

COMMENT

When this and the two other cases of amaurosis were first seen I was very doubtful as to

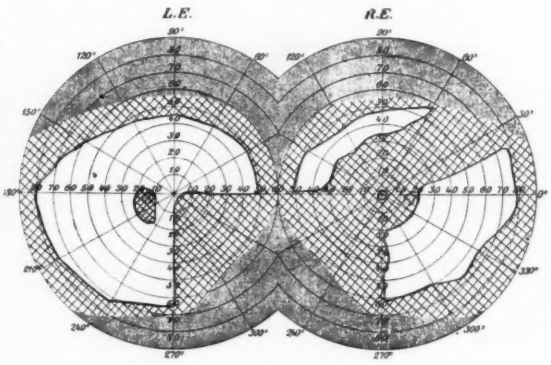

Fig. 4
Visual fields of Case W. N. on May 5, 1931

the exact location of the lesion causing the blindness. Sudden blindness affecting both eyes simultaneously is unusual unless due to some disturbance or lesion affecting the visual areas of both occipital lobes. Such a thing we see in the amaurosis which occurs rarely as a complication of uremia. A collection of pus between the posterior portions of the occipital lobes might, by pressure or the infiltration of toxins, affect both visual areas simultaneously.

A certain amount of encephalitis is known to occur in all severe purulent meningitis. According to Wertham,[5] apart from the encephalitis and toxic-degenerative changes, other extensive lesions of the nervous parenchyma occur. The cortex is the part chiefly involved, but the subcortical structures, such as the basal ganglia, may also be severely affected. However, if the lesion were confined to the central visual areas, the pupillary reaction to light should be present, but in each one of these cases it was absent. Furthermore, in the case reported above, optic atrophy developed later, therefore the lesion must have been anterior to the primary optic centers. Another point against the lesion's being in the cortex is found in the fields. Examination of these show that the quadrant defects do not represent corresponding parts of the retina such as would result from a lesion posterior to the chiasm, but indicate rather that the lesion is in the chiasm itself.

Another point of interest is why central vision recovered first and more completely than peripheral. This undoubtedly was due to the anatomic arrangement of the nerve fibers in the optic nerve and chiasm. In the posterior portion of the optic nerve and in the chiasm the papillomacular bundle is centrally located and therefore was not subjected to the same degree and intensity of damage as the fibers from the peripheral portions of the retina.

It seems, therefore, that we are forced to the conclusion that these cases of sudden and complete blindness, occurring as a complication of meningococcic meningitis, were due to lesions of the optic chiasm, probably an intense neuritis.

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Exchange Building

DISCUSSION (*Abstract*)

Dr. F. H. Clements, Birmingham, Ala.—The author has reported only the affections of the optic nerves or visual pathways. It would be interesting to know if, in his series of 184 cases, there was any involvement of the intrinsic or extrinsic muscles of the eye. Mydriasis, myosis and paralysis of the sixth and eighth nerves are reported as occurring in varying percentages in other series of cases.

As to the cause of the neuritis with elevation of the disc, we should consider two factors, acting singly, or together:

(1) Increased intracranial pressure; and

(2) Inflammation of the optic nerves.

To support the increased intracranial pressure theory, we have the following:

(1) The vision is not usually impaired.

(2) The appearance of the optic nerve more often resembles the pressure type of choked disc, with very little involvement of the surrounding retina and with no exudate present.

(3) The best treatment for the elevated discs is spinal puncture to relieve the intracranial pressure.

To support the inflammatory theory, we have the following:

(1) Some cases show a typical inflammatory type of choke disc with involvement of the surrounding retina, and with much exudate and hemorrhages.

(2) The disturbance of vision and the field defects found after recovery in a few cases are more than you would expect from simple increased pressure which is being relieved by lumbar punctures during the course of the disease.

It is probable that both of these factors enter into some cases, but I do not believe that the effect of increased intracranial pressure is given as sufficient importance as it deserves. I should like to hear this point discussed.

The nature of the inflammatory lesion, according to Terrien, is a diffuse optic perineuritis extending along the optic nerves or tracts, with some involvement of the nerve tissue itself at points of intense inflammation, causing an interstitial neuritis. This interstitial neuritis is responsible for the defective vision and the subsequent optic atrophy, as shown by the field defects. I agree with the author in his conclusions as to the location and nature of the lesion causing amaurosis.

As the author points out, it is very surprising that more involvement of the cranial nerves does not occur in cerebrospinal meningitis, considering that they are continually bathed in a puddle of pus at the base of the brain.

Dr. R. O. Rychener, Memphis, Tenn.—Dr. Lewis in this paper and in the previous report presented before this Section in 1928 discussed a subject which has been touched but slightly in medical literature.

The explanations which the text books give for the amaurosis of meningococcic meningitis are surprisingly meager, and it is further surprising that autopsy reports have added little to our knowledge of this phase. I am inclined to believe with the author that amaurosis results from an acute toxemia of the optic nerves, chiasm or primary optic centers due to an adjacent meningitic process. In the light of the recent advances in neurosurgery and the recovery of vision following the breaking up of scar tissue about the optic nerves in cases of blindness due to localized arachnoiditis,

might it not add to our knowledge to open and inspect through the transfrontal route some of these totally blind cases after all symptoms of active meningitis have subsided? This procedure in good hands can be safely advised and some means of therapy may thereby be discovered.

Dr. Lewis (closing).—We saw very few muscle paralyses in our series, about six or eight cases, I think.

The neuritis seen in these cases is a diffuse interstitial affair. I have had the opportunity of examining only one of these nerves histologically. The fibers were swollen and the entire nerve infiltrated throughout with leucocytes, mostly of the polymorphonuclear variety.

ADJUSTABLE SHOULDER FOR PARACENTESIS NEEDLE*

By EDWARD LEWIS, M.D.,
Washington, D. C.

There are instances in which an adjustable shoulder on a paracentesis needle is of value.

Such a device is useful in cases in which it is desirable not to exceed a certain degree of penetration, as in puncture of the superior longitudinal sinus or the cisterna magna. It is also useful in fixing the degree of penetration in secondary operations after an initial one in which the proper depth has been determined; and in fixing the depth to which the needle may go, once that depth has been attained at a given operation, as in paracentesis thoracis, where sudden lurching on the part of the patient, especially in the case of a child, might drive the needle to dangerous depths.

The adaptation to this purpose of the ordinary metal scarfpin guard or clutch, put on the needle in a position the reverse to that for which it was originally intended, gives a device which is simple, useful, inexpensive, easily obtained, and easily sterilized.

1801 Eye Street, Northwest

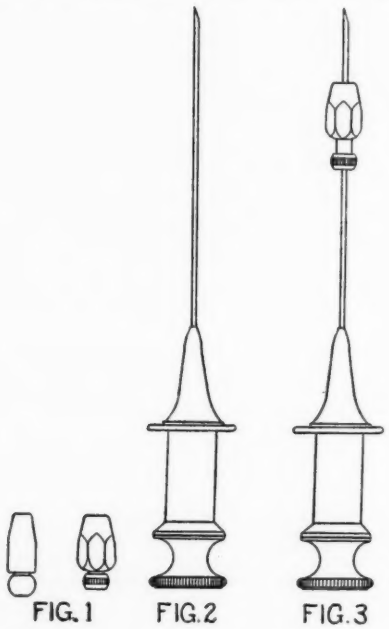

Fig. 1. Types of scarfpin guard or clutch.
Fig. 2. Paracentesis needle.
Fig. 3. Adaptation of guard as adjustable shoulder on needle. Note the reverse position.

*Received for publication January 2, 1933.

ABORTIONS*

By WM. T. MCCONNELL, M.D.,
LOUISVILLE, KY.

A miscarriage, considered from a medical standpoint, includes all premature emptying of the contents of the pregnant uterus from the time of conception to viability. The terms abortion and miscarriage, in a scientific sense, are synonymous, and will be used in this discussion interchangeably. Before entering upon the matter of diagnosis and treatment, it might be well to consider the underlying principles that bring about miscarriage. These factors may be grouped under four general heads as follows: (1) abnormalities; (2) infections; (3) trauma; and (4) toxins.

Abortions in the early months of pregnancy are practically always preceded by the death of the fetus so that the consideration of this type resolves itself into the discussion of those factors which produce fetal death. In the later months of pregnancy, however, the fetus is often born alive and other causes must be taken into consideration. In many cases there is a combination of circumstances entering into the production of abortion so that more than one of the above named factors play a part.

One of the most usual causes of death of the fetus is abnormality in fetal development. These abnormalities would, if pregnancy persisted, produce monstrosities. Fortunately most of these

*Read in Section on Obstetrics, Southern Medical Association, Twenty-Sixth Annual Meeting, Birmingham, Alabama, November 15-18, 1932.

terminate in early abortion. There seems to be no adequate explanation of the condition.

A second form of abnormality is found in the appendages of the fetus such as torsion of the cord, hydramnios, hydatiform or blood moles. Other conditions in this group are hypertrophic endometritis, abnormalities of the placenta, such as infarcts, hemorrhagic areas of the placenta or the decidua, or atrophic conditions of these organs. In these conditions the nutrition of the embryo is interfered with to such an extent that it perishes from lack of oxidation or nutrition.

Placenta previa, premature separation, marginal implantation of the cord are all formidable factors in abortion. These abnormalities usually produce their evil effects in the later months of pregnancy.

Abnormalities of the uterus are also factors which play an important role. Among these may be mentioned the undeveloped uterus, fibroids, uterine displacements and hypersensitiveness of the uterus. In the undeveloped uterus pregnancy rarely takes place, but when it does the normal uterine hypertrophy is not sufficiently rapid to nourish the fetus and intrauterine death occurs.

Fibroids do not as a rule impair or endanger pregnancy unless by their situation or size they rob the placenta of its needed amount of oxygen and nourishment or unless their location is such that a mechanical interference with fetal development ensues. Uterine displacements usually produce abortion by causing a poverty of nourishment to the fetus due to interference in the uterine circulation produced by the displacement. In some cases, however, where the uterus is firmly adherent in a retroversion the abortion is produced by more mechanical means.

Some uteri that are apparently otherwise normal are found to be hypersensitive to external stimulation. Such a uterus is probably faulty from a developmental standpoint. This condition may be produced by disease of the cervix, endometrium or myometrium, but is sometimes encountered in patients in whom no such diseased condition can be demonstrated. In a hypersensitiveness due to disease the patient usually aborts at about the same time in each succeeding gestation, but in the type of hypersensitiveness that is not accompanied nor caused by disease a patient may carry several succeeding pregnancies, each one somewhat longer, until finally a living child is the result.

Among the factors causing abortion, infections probably play the greatest role. They may be divided into acute and chronic. Among the acute infections may be named typhoid fever, pneumonia and other diseases of this type. The production of abortion in these cases is probably due to the toxin produced by the disease which acts upon the fetus in such a way as to cause its death. Abortions of this type may be considered under the heading of those due to toxins. In chronic infections, nephritis, myocarditis, endometritis and cervicitis are the most frequent causative factors. In syphilis, abortion is usually late in pregnancy and is caused by the hypertrophic condition of the placental blood vessels and villae with a resultant diminution in the nourishment of the fetus. In nephritis, the toxins of the body are the most probable disturbing element. In myocarditis, improper oxidation of the blood, together with toxins, would account for the abortion. Endometritis may be acute or chronic, the acute type usually being encountered in criminal abortions and the chronic type often having for its ground work a pre-existing Neisserian infection. The decidua in these cases become engorged and hemorrhagic and not only is the nourishment of the fetus impaired, but also the toxins produced by this condition militate against its existence.

The writer has seen a considerable number of cases in the last few years in which endocervicitis was the only abnormal condition discovered in a case of miscarriage. This condition is often associated with endometritis, but very frequently we have found either that the uterus was free from infection or the cervix was the chief offender.

At the present time we are watching with a great deal of interest the effects of infection due to the *Bacillus melitensis,* the organism causing Malta fever. It is well known that in the porcine and bovine types abortion is the most outstanding characteristic in animals, but in the human there is not enough data as yet upon which to draw conclusions. Some cases in which the patient was suffering from this disease have terminated in abortion, but whether or not the specific organism was entirely responsible has not been determined.

As to trauma, the normal pregnant uterus will withstand considerable shock without apparent detriment to the fetus, but where there is a hypersensitiveness such things as jolts, falls or sudden exertion are often sufficient to bring about premature emptying of the uterus. In some cases the hypersensitiveness is so great that digital examination or coitus is sufficient to produce the abortion. In other instances it seems almost impossible to produce abortion by

trauma. Many cases go on to full term even after dilatation of the cervix and the insertion of foreign substances.

Toxins play a minor role in abortion, if we except the toxins of acute or chronic infections. Such agents as lead, gas and similar poisonous materials are sometimes sufficient to terminate pregnancy.

Under the head of toxins may be mentioned endocrine disturbances. Certain individuals are afflicted with hyper- or hyposecretion of some particular ductless gland. These disturbances, when sufficiently marked to interfere with pregnancy, usually prevent conception. At times, however, the endocrine dysfunction seems to be aggravated by the occurrence of pregnancy and abortion follows. In thyroid disturbances the production of an excess of thyroxin usually prevents pregnancy, but if this hypersecretion occurs after conception has become established, it may result in premature termination of pregnancy. This is also true of hypersecretion of the pituitary gland. The dysfunction of the ovaries and especially of the corpus luteum more frequently results in metabolic upsets than in abortion. The interdependence of endocrine functions renders it particularly difficult to determine which gland is at fault primarily.

From a clinical standpoint miscarriages may be classified as spontaneous or induced. Those that are induced may be therapeutic or criminal. From a standpoint of treatment they may be threatened, inevitable, incomplete or complete.

As to diagnosis of a threatened miscarriage, the factors that would lead us to suspect the likelihood of abortion are, first, bleeding; and second, cramps. Many patients bleed at irregular intervals during pregnancy, but carry the fetus to term. But bleeding in the early months of pregnancy is always a rather serious complication and should put us on our guard. Especially is this true when it is associated with cramps. The character of bleeding may or may not be of help in determining whether or not miscarriage is threatened. If the fetus has died there is usually a discharge of dark brown or grumous material, usually having considerable odor and persistence. This may go on for many days before the uterus empties itself. The cervix in this condition is usually not dilated; neither has it become shortened.

In the inevitable type of abortion the cervix becomes somewhat dilated and shortened and the bleeding is usually more severe and practically always accompanied by cramps.

In an incomplete abortion the fetus is usually passed with a part or all of the placental tissue still remaining within the uterus. If it is known that the fetus has been discharged and bleeding is profuse, if cramps persist or if placental tissue can be felt in or through the cervix, then the condition is referred to as incomplete.

A complete abortion occurs when all of the products of conception except the decidua are expelled. When the abortion is complete, cramping is practically always discontinued, the bleeding is not profuse and the cervix regains its tone. Before discussing the active treatment of miscarriage it will not be amiss to consider prophylaxis.

The prevention of abortion resolves itself into discovering the etiological factors concerned and correcting them. In acute infections the principal measures should be directed toward relieving the general condition. In chronic infections our greatest hope lies in preventing subsequent abortions. In syphilis it has been clearly demonstrated that adequate treatment, including the arsenicals, gives very happy results, especially if given early in pregnancy. In myocarditis a strengthening of the heart muscle by appropriate remedies and adequate rest produces good results. In chronic endometritis a curette may be of great value; in cervicitis and endocervicitis the active treatment of infections of the cervix by electrocautery or by other forms of surgery is indicated.

Foci of infection in the tubes should be eliminated, as should any other more remote chronic disease. Where some abnormality exists which is remediable, the procedure is obvious. The removal of fibroids, the replacing of retroversions, the correcting of prolapses can be done in the interval between pregnancies.

The hypersensitive uterus can be dealt with by removing all sources of irritation. The avoidance of all unnecessary exertion, rest in bed, sedatives and a close watch for signs of threatening abortion will often meet with a successful termination.

When abortion is due to endocrine disorder, in which some one gland is definitely and clearly at fault, benefit may be obtained from ductless gland therapy; unless, however, the condition is clear-cut,much harm may be done by experimenting with these powerful agents. The treatment of such conditions is as yet in a more or less experimental stage and no definite rule can be laid down for its application.

In the treatment of threatened miscarriage, the patient should be kept in bed, the foot of

the bed elevated, all disturbing conditions should be removed, opiates and sedatives should be freely employed. Some writers advise the use of epinephrine because of its relaxing influence upon the uterus. The patient should be watched carefully for signs of hemorrhage and for systemic complications. If the bleeding becomes very profuse, interference may be necessary, although according to the accepted standards the miscarriage may merely be threatened.

As a general rule, the same line of treatment will apply to cases of inevitable abortion, unless hemorrhage becomes severe, or unless fever is present. If there is any reason to hasten the termination of the condition, pituitrin given in 0.5 c. c. doses at half-hour intervals for six doses, followed by fluid extract of ergot 1 dram every 4 hours for 3 doses will usually be sufficient to complete the process. At any rate, the patient should be treated expectantly, unless severe bleeding or her general condition make interference necessary.

Our greatest problems in the treatment of miscarriages are naturally encountered in those that are incomplete. Methods of handling these cases range all the way from curettage of all cases to strict non-interference, no matter what happens. The writer has known men who would clean out every uterus in every case of miscarriage, whether the abortion was inevitable, incomplete or complete, regardless of attending circumstances. This practice is mentioned only to be condemned.

In outlining any course of treatment for incomplete abortion the procedure must be a contingent one. My discussion, therefore, will be a recital of what appeals to me as the most sensible course in the various complications usually encountered.

It is gratifying to observe that the general trend is toward conservatism. The profession seems to be coming to a realization that ill-advised radicalism is too frequently fraught with dire results.

In this connection we would like to submit the general plan of treatment of the Department of Gynecology of the City Hospital as corresponding in the main with the writer's views. The following outline was taken from a report from that Department, read before the Louisville Obstetrical and Gynecological Society by Dr. W. O. Johnson:

"After the patient enters the hospital, energetic systemic treatment is instituted at once in the form of absolute rest, fresh air, and forced feeding of a high caloric, low residue diet. The fluid intake is kept up to 2000 c.c. per day or hypodermoclysis may be resorted to and blood transfusion is given without hesitancy if dessication or anemia is present.

"The initial examination is made very cautiously and if there is definite evidence of complication outside of the uterus, even so slight an indication as abdominal tenderness, strict conservation is observed, and the following treatment is directed towards the resulting complications and not abortion.

"If upon speculum examination, under sterile precautions, we see remnants protruding from the dilated cervical os, they are gently removed with placenta forceps. The cases are started on 0.5 c.c. of pituitrin each half hour for six doses and at the completion of this course 1 dram of fluid extract of ergot is given every 4 hours 4 times. Under this regime a severe hemorrhage very seldom occurs, and it is rarely necessary to pack the vagina. In fact, the vagina is never packed except in rare and extreme cases of profuse bleeding, and this only long enough to tide over an acute emergency until the pituitrin and ergot have acted. Even if the bleeding continues and the membranes are not completely expelled, a dilatation and evacuation is not considered until the temperature has been normal for three days, and then under the most rigid aseptic operative technic.

"Hegar dilators are used to dilate the cervix, and with an iodine sponge wrapped on dressing forcep the uterine cavity is swabbed out. With sterile gloved finger the uterine cavity can usually be examined with ease to detect any remnant of tissue. Forceful dilatation and rough handling with hands are contra-indicated. After dilatation and evacuation the uterus is kept in contraction by ergot, and iodine sponge is left in the cervix for 24 hours."

The writer has encountered a number of cases where the retained placental tissue was very adherent to the uterine wall. In these cases the sharp curette has seemed to us to be the best instrument to use. Great care must be employed, as much harm may be done. Gentleness of manipulation is absolutely necessary, or the uterine wall may be perforated. After the completion of the operation, the uterine cavity is packed snugly with a narrow strip of iodoform gauze, and the vagina is packed loosely with a 2-inch sterile gauze strip.

Conservative treatment in the presence of infection should be strongly emphasized. Very rarely, indeed, would curettage be justifiable in this condition. Usually, such a procedure tends to convert a local disturbance into a generalized sepsis. Where considerable blood has been lost, it is very important to build up the general condition with such things as intravenous saline, glucose, transfusions, and other supportive measures.

In the aforementioned report Dr. Johnson reviews 288 cases of incomplete abortions on the City Hospital service treated by the routine outlined.

There was not a death from dilatation and

curettage, and only in 10 per cent of all these cases was this curettage necessary. There were 3 deaths in this series or a mortality of 1.4 per cent. Two deaths occurred from septicemia and one patient entering the hospital almost moribund died from hemorrhage. Of the 288 patients, 63 gave a history of inducing the abortion and 25 more admitted operations by criminal abortionists. The other causes given were as follows: fall, jar or other strain, 46; medicines, 29; catheter, 23; malposition, 23; unknown, 96. There is no way of determining the exact number of criminal abortions in any series reported because the history is not always reliable. Even with the facts that were ascertained, the mortality rate was exceptionally low, which is an eloquent argument in favor of conservative treatment.

The ratio of miscarriages to viable births is given by various authors as anywhere from 1 in 3 to 1 in 5. These estimates are probably very conservative. Many states have no law requiring the reporting of these cases, so no definite data are possible.

Ignoring those intentionally induced, the number is still of sufficient magnitude to constitute a medical and social problem. The social integrity of a race depends largely upon its reproductiveness. Every miscarriage militates to weaken this integrity.

From a medical standpoint, every uninduced abortion signifies some physical weakness or disease, and our responsibility as physicians does not end with the termination of the miscarriage. These cases should, on the contrary, afford us opportunity for investigation of the general condition of the patient and the discovery and correction of defects which otherwise would remain unrecognized.

One of the darkest blots upon our modern civilization is the criminal abortionist. Until the medical profession finds some effective means of ridding itself of this baneful member, it must bear the stigma or its share of responsibility for the perpetuation of the evil.

When a man takes another human life in the heat of anger, or in self-defense, or for the protection of others, it is, to say the least, a deplorable occurrence. But we might find grounds for condonement. The highwayman who kills for gain is condemned by society as one who has forfeited his right to live. But even he operates against those who have some powers of self-defense.

But the criminal abortionist, for the proverbial thirty pieces of silver, or less, will destroy a human life which is utterly defenseless in a case in which there can be no plea of heated passion, no basis for self-defense, a life that cannot even raise the cry of the new born infant in its own defense. Nor is this all. The yearly toll of mothers' lives taken by those doctors of medicine is a matter of no small amount. And these men hide behind the cloak of membership in the honored and sacred medical profession.

Why are these malefactors seldom brought to justice? Why is it difficult to secure a conviction when one of them is indicted? Why are few of them deprived of the license to practice medicine? It seems to us that there is a work for us to do, a task that is not being done as it might be. It could not be said of us that we are not worthy as an organized body to cast the first stone.

The women who patronize these men are not without blame, and yet their offense is far less culpable. In the early weeks of pregnancy the woman is often the victim of a mental depression. She lives under a cloud of fear and dread that amounts in many cases to a psychosis. If she can be safely carried past this state by the wise and kindly physician, he will later have her undying gratitude. Here again we can do much toward mitigating this evil. The laity should be better informed as to the dangers of such a procedure as well as the moral phase. It is our office to give this information.

One of the most disagreeable tasks of the conscientious physician is to care for these patients after the abortionist has done his work. He must assume an unfair responsibility. When it can be ascertained that the abortion has been illegally induced, it is the attending physician's duty to himself to acquaint the patient and other responsible members of the family of the danger and eventualities in the case and let it be definitely understood where his responsibility ends.

In conclusion, the whole subject of miscarriage and abortion is one which greatly concerns us as a profession. No phase of our work partakes more fully of the medical, moral and social.

It is our responsibility primarily to give immediate care to those who place themselves in our hands and to treat with conservative thoroughness the problem at hand. Then the more remote factors which brought about the condition deserve our most careful consideration, with the end in view that each patient may be returned to normal health.

Finally, our obligation reaches to the correction, as far as lay in our power, of the social evils following in the wake of criminal procedures.

DISCUSSION (*Abstract*)

Dr. W. O. Johnson, Louisville, Ky.—I wish to emphasize some of the points which Dr. McConnell has made, as follows:

(1) The physician has a great responsibility to those patients who have repeated abortions, because of local or constitutional conditions. They are indeed problems. Most of them are badly in need of proper treatment, not only for the betterment of their future health, but also to enable them to have normal pregnancies.

(2) In most instances of falls or traumata that are followed by abortions, I believe the trauma is coincidental. The abortions are usually the result of defective germ plasma, improperly developed or diseased placenta or uteri. The abortion would inevitably have occurred without the trauma.

(3) Chronic endocervicitis and its resulting uterine changes as a remote cause of abortions cannot be too strongly emphasized. Such conditions can be greatly benefited if obstetricians will inspect all of the pelves six to eight weeks after delivery, and treat with the cautery or have corrected by operation the infections or pelvic injuries that arise from the delivery.

(4) My experience with hyperthyroidism does not coincide with that cited by Dr. McConnell. Many cases of hyperthyroidism, before their vitality has been depleted, conceive readily, and carry their pregnancies to full term without much disturbance. In the majority of these cases, however, there is an exacerbation of acute hyperthyroidism two weeks after the delivery. Very careful handling and iodine prophylaxis are of great importance in these cases.

(5) Corpus luteum disturbances render patients less able to conceive, and causes early abortions once conception does take place.

(6) To re-emphasize the increasing importance of this subject which Dr. McConnell has ably presented, may I present some recent statistics from the Gynecological Department of the Louisville City Hospital?

During the past four years we have had a yearly average of 10,733 patients admitted to the Hospital with a yearly average of 191 cases of abortion: an average of 10.7 per cent of admissions to the Hospital are threatened or complete abortions. These cases have had an average of eight days of symptoms before admission to the Hospital.

In over 50 per cent of these cases, different procedures have been used to bring on the abortion, and in 8 per cent of the cases they are known to have been induced by a physician.

If we divide these cases into three age groups, as follows:

13 to 20 years of age	22.4 per cent of cases
20 to 30 years of age	50.2 per cent of cases
30 years of age or over	27.3 per cent of cases

This is an average of cases according to groups from September, 1928, to September, 1931.

From September, 1931, to September, 1932, there has been an increase of 0.2 per cent in the number of cases of abortions admitted, and an increase of 7.6 per cent in the number of cases in the 20 30 year group, with a greater percentage of increase in the multiparous women who, because of numerous circumstances, have induced their abortions.

This portrays the gravity of the problem at hand, but it does not touch the surface of the long train of complications, prolonged ill health and deaths that follow in the wake of these cases.

Dr. McConnell has clearly pointed out the responsibility of the profession to give proper immediate care to these cases, and to treat them with conservative thoroughness to determine and correct, if possible, the direct and remote factors which may bring about such conditions, and to eradicate the professional menace by education of the public to know the grave dangers and complications and death that result from induced abortions.

Dr. J. M. Nokes, University, Va.—We have never been entirely familiar with the true causes of abortions. There have been many etiological factors suggested, which should be corrected if possible, with the hope of avoiding recurrence.

During the past few years we have noticed a definite increase in the incidence of abortions, particularly among the private class of patients. It has been easier to elicit positive histories of criminal intervention than formerly. We have observed repeated abortions in cases of glandular dysfunction in which the patients do not show any gross evidences of underdevelopment of the internal genitalia. In this type of patient after the proper glandular therapy was instituted pregnancy would proceed to term. In the last 100 abortions observed, we have obtained two positive agglutinations with *B. abortus* but have been unable to check up on the home surroundings of the patients satisfactorily enough to warrant any deductions. Occasionally we have seen a tendency toward repeated abortions in certain of the blood dyscrasias.

In the treatment of abortions, where the patient has been observed during several pregnancies and has shown a predisposition toward abortion, existing pathological conditions should be corrected. Any stimulus which might possibly serve as an exciting cause of abortion should be removed.

I think we are too prone to make a diagnosis of threatened abortion and occasionally overlook an extrauterine pregnancy. It may be that by examining routinely all cases of early pregnancy associated with bleeding we precipitate an inevitable abortion, but it is safer to do this than to fail in the detection of an ectopic pregnancy. The dangers from infection after a carefully done sterile, vaginal examination are comparatively slight.

When to interfere with a case of threatened abortion has long been debatable. The pains usually respond to sedation and ice caps, while the bleeding continues. If there is little or no improvement in the condition of the patient after five to seven days of conservative treatment, I think we are justified in emptying the uterus by the most conservative means available. I cannot see the sense of allowing the patient to get up and about for several days or weeks in the hope that the abortion will become inevitable. The more cases of spontaneous, threatened abortion I see the more convinced I am that nature is attempting to expel an undesirable product. I believe that a soft rubber catheter and cervical pack inserted in the cervix, and tight packs in the cul de sac for counter irritation should be the method of choice. This procedure should be preceded by a hot soap and water enema and quinine

and followed by fractional pituitrin (0.5 c.c. each half hour for 6 doses) beginning two hours after the packs and catheter have been inserted.

If the abortion is complete there is no need for further interference. The physician should satisfy himself that the entire product of conception has been expelled. If a portion has been retained there is no need for immediate interference unless the bleeding is excessive as often the administration of fractional pituitrin will produce sufficient contractions of the uterus to expel any remaining fragments. If the bleeding becomes alarming a careful examination should be made and any remaining portions of the placenta removed by digital curettage or sponge forceps.

In cases of infected abortions I feel that a gentle, sterile vaginal examination should be made at once unless the physician is sure that complete abortion has occurred. Often we see a patient for the first time with a history of abortion, an elevated temperature and pulse and upon examination find a large piece of necrotic placenta protruding from the external os of the cervix. The gentle removal of this tissue by fingers or sponge forceps often brings about a rapid improvement in the clinical picture. There is absolutely no excuse for the use of the curette in this condition. Other than this, the treatment of infected abortions is the treatment of sepsis in general with special emphasis on repeated blood transfusions rather than sera and blood stream antiseptics.

PERIODIC EXAMINATIONS*

By W. N. Blount, M.D.,
Laurel, Miss.

The public as a rule thinks of a railroad surgeon only as one whose duty it is to take care of persons injured by the railroad. As a matter of fact, this is, as it should be, only a small part of his duties. The safety campaigns inaugurated a few years back by the different railroads have resulted in reduction of injuries by 50 per cent and in some instances even more. This has been done, not by accident, but by systematic teaching of employes the danger points in their occupations, constant warning by their foremen to be careful, and by the introduction of safety appliances for their protection.

Another great factor in this reduction of injuries is the watchful care given the employes by the medical department of every railroad company in keeping check on and correcting their physical and mental diseases. This is being done by practically every railroad company by having periodic examinations and keeping records of the physical condition of every employe. The health of the employe is a valuable asset to any corporation and deserves the same time, expense and care to keep it normal as any piece of expensive machinery necessary for the successful operation of that particular industry.

We know that a train never leaves a terminal point until it has been thoroughly inspected by one trained in that line of work, from the engine on down to the last coach, if it is a passenger train, or to the caboose if it is a freight train. What about the engineer, who is one of the most important parts of that train? How long has it been since he was okeyed by a medical inspector? We have records of appalling loss of life following the failure of man power at a critical time. This does not count the tremendous monetary loss caused by a momentary lapse of memory of the engineer, if he forgets his train orders, or thinks it not necessary for him to follow orders, knowing that he can make a certain point before meeting an oncoming train, and failing. Or a sudden heart attack may render him lifeless, with possibly hundreds of lives behind him depending on his alertness and physical well being.

There is another phase. We owe it to the employe as a bread winner of the family to keep him in as perfect health as possible in order to increase his earning power, enable him to support his family better and make better citizens of his children as they grow to manhood and womanhood.

We, as representatives of the medical department of our respective companies, can play an important part in bettering the health condition of our employes and preventing possible accidents to them and to others by encouraging periodic health examinations. To do this it is necessary to use diplomacy in gaining the confidence of employes, for many will think that this is nothing but an excuse to take them out of the service. By tactful handling, however, we shall gain their confidence and find our services appreciated. We see them coming back of their own accord asking that we check them over instead of objecting, as at first.

My own personal experience when we began making periodic examinations on our lines was that a considerable unrest developed among the employes, especially in the transportation department. I have made it a rule never to begin an examination of anybody until I have explained to the employe the purpose of the examination, assuring him that it is not our purpose to remove any one from the service; that the examination is only a safety measure to protect the health of the employes; that if any-

*Read in Section on Railway Surgery, Southern Medical Association, Twenty-Sixth Annual Meeting, Birmingham, Alabama, November 15-18, 1932.

thing is found wrong he will be told what it is and what is necessary to do towards correcting the trouble. I am happy to say that no one has been removed from the service as a result of the examination, and I am sure many were benefited by the advice given. All who were found to have any trouble that later might interfere with their ability to continue working were advised to report at regular intervals for observation. It was a pleasure to hear them express, when reporting for these check-ups, their appreciation of the interest shown in our efforts to keep them on the job. They oftentimes said they were not aware that anything was wrong until it was discovered at this periodic examination.

In these examinations will be found many unsuspected local foci of infection which may cause various diseases such as heart lesions, hypertension, kidney involvements, and so forth. If they are treated, many years of usefulness may be added to the employe's life. If they are not corrected, his physical condition may get beyond repair.

We, as company surgeons, play two very important roles, one as guardian of the health of the employes, the other as protector of the interests of our respective lines, and we should accept the duties in all seriousness. It is a big job.

We should encourage every employe to take advantage of our services in order that he may keep fit and be better able to perform his duties and to provide for his dependents. We owe it to our respective companies, if we find an employe suffering with a defect that might render him a hazard to his fellow employes, the company or to the traveling public, to notify the proper official of the condition found in that employe, so that necessary steps may be taken to protect all concerned.

Only by periodic examinations may we discover beginning diseases that can be corrected by treatment before they reach that stage when it might be necessary to take a man out of the service, a very disagreeable duty.

The Committee of the American Railway Association, Medical and Surgical Section on Developments Resulting from Periodic Physical Examinations, of which I have the honor of being a member, in the early part of the year sent out a questionnaire to the members of the Association. The Chairman of this Committee, Dr. S. B. Taylor, of the New York Central Lines, in making his report to the Association, which met in New York in June, said:

"We want to compliment the Representatives in the Section on giving our questionnaire more consideration in answering the questions than they have shown heretofore. This added interest bespeaks well for the advancement of periodic examinations.

"After contacting medical officers who have had charge of periodic examinations in various railroads in the country, we are now able to report a decrease in focal infection and their sequelae. We are further able to report that there has been a much earlier discovery of disqualifying defects accountable to syphilis, with the result of early, intensive treatment, which will no doubt lessen the number of paretics, tabetics, and so forth.

"There is no question that the periodic examination has come to stay. Even if the railroads felt themselves too poor to stand the expense, I believe the men would insist upon taking it upon their own shou'ders, as they are now relying on this examination to keep them healthy and fit. There has been a noted increase in efficiency in departments that have been under close observation of the medical directors and chief surgeons through the periodic examinations."

In conclusion, I am quoting another member of this Committee, Dr. J. C. Landengereger, Chief Surgeon of the Oregon Short Line Railroad, who says:

"In the periodic examinations we are dealing with members of our own railroad family, men who have certain rights in the family, men upon whose shoulders rests the well-being of our passenger patrons as well as the physical property of the railroad company, and in whom the company expects its physical examiners to make every effort to detect all possible symptoms pointing toward hazard and physical failure."

Hotel Pinehurst

DISCUSSION (*Abstract*)

Dr. Edward T. Newell, Chattanooga, Tenn.—There is no more important action that the railroads could take than to establish periodic medical examinations of the employes. There are many instances on record where engineers have dropped dead at the throttle; where street car conductors have been stricken with paralysis or other illness. Had a previous examination been made, a catastrophe might have been averted and the common carriers for which they worked might have been spared the expense incident to the accident.

The Brotherhood of Railway Locomotive Engineers, a very strong and intelligent organization, is heartily in favor of these examinations, and I trust that the time will come when the railways, the unions and all the men employed by railroads throughout this country will demand them.

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EDITORIAL DEPARTMENT

SOUTHERN MEDICAL ASSOCIATION
Twenty-Seventh Annual Meeting
RICHMOND, VIRGINIA, NOVEMBER 14-17,
and a post-meeting day in Washington, November 18

RICHMOND IN NOVEMBER

As the Editor of the Richmond News-Leader says elsewhere in this issue,[1] describing "Richmond's Confederate Epoch,"

"Quebec, New York, St. Augustine, New Orleans and Austin recall international rivalries for the possession of America; Richmond is the emblem of the one conflict that threatened the unity that had been achieved after those rivalries had been settled."

The historic importance of Richmond antedates the Civil War and even the American Revolution. It was in Richmond that Patrick Henry made his famous "Liberty or Death" address. Richmond is more famous as the home of John Marshall, the most distinguished jurist of American history. Marshall was Chief Justice of the Supreme Court, and he held that position for thirty-four years, from 1801 to 1835. Through his early interpretations of the young constitution he had a greater influence than its authors, or than the presidents in directing the course of the new republic. He laid down in 1803, for the first time,[2] the doctrine not expressly conferred on the Court, of the supremacy of the judiciary: that the judges have the power to declare an act of Congress null and void when in their opinion it violates the constitution. He set aside important acts of state legislatures, although the legislatures themselves passed sheaves of resolutions protesting and condemning his decisions. From his day, by his precedent, the authority of Federal and state courts to pass upon the constitutionality of laws has remained unshaken, and the tradition established by him has remained sacred.

Modern Richmond does not live on the reputation of by-gone glory, but according to the distinguished old tradition. There is no more progressive city medically than the Richmond of today.

The plans for the local clinical sessions of the opening day of the meeting, the general sessions, the section and special programs, are in shape to indicate that the best medical pabulum will be provided for the twenty-seventh annual meeting of the Association in November. Widely known physicians from the South and from many parts of the country will sift the achievements of the year in general medicine, surgery, and the specialties. The details of the program, meeting places and general arrangements will appear in a later issue. The railroads will offer special rates. Hotel reservations should be made early.

1. Freeman, Douglas S.: Richmond's Confederate Epoch, this issue, page 746.

2. Beard, Charles A. and Mary R.: The Rise of American Civilization. New York: Macmillan Co., 1927.

FURTHER STUDIES ON EXTRACTS FOR TREATMENT OF PERNICIOUS ANEMIA

If one were asked to name the outstanding clinical advance of the past decade, since the discovery of insulin, it is probable that the control of pernicious anemia with liver and liver extract would be thought of first. Here a disease known for years and accepted as incurable came under the medical thumb; a specific treatment was shown. A great many workers deserve credit for the discovery, and still more have advanced the studies since the first demonstration that remissions could be effected by diet. Whipple and Robscheit-Robbins,[1] of Rochester, showed that hemoglobin regeneration after severe bleeding of dogs depended upon the diet and was stimulated by liver more than by other foods. Surprising as it seems, the effect of diet on the blood picture had received little medical atten-

1. Whipple, G. H. M.: and Robscheit-Robbins, F. S.: Blood Regeneration in Severe Anemia. Amer Jour. Physiol., 72:395, 408, 419, 431, 1925; 79:260, 271, 280, 1927.

tion up to that time. Minot and Murphy,[1] of Boston, in 1926, demonstrated remissions in human cases of pernicious anemia following diets which contained a large quantity of liver. Castle[2] and associates with lean meat digested in a normal stomach, and Sturgis and Isaacs[3] with dried stomach mucosa, likewise stimulated the formation of reticulocytes and produced remissions in symptoms of pernicious anemia. The Harvard group[2] called pernicious anemia a protein deficiency disease. They believe that it is caused by failure to utilize protein products and compare it to diabetes, a disease of carbohydrate deficiency, or failure to utilize carbohydrates. More recently Roger Morris[4] and his associates at the University of Cincinnati have achieved excellent results by injecting intramuscularly normal gastric juice which has been neutralized and concentrated. The stomach secretion of normal persons is inactive after oral administration to pernicious anemia patients. Morris' concentrate, however, after intramuscular injection, produced such a rapidly favorable effect upon the blood picture that he refers to it as the hematopoietic hormone of the stomach. He even postulates a clinical condition of hormone insufficiency which is pernicious anemia; and another of hormone over-activity, which is erythremia.

Helmer, Fouts and Zerfas,[5] of the Lilly Laboratory and the University of Indiana School of Medicine, have attempted to pyramid the liver and stomach effects, to obtain a still more potent red cell stimulant which would be active by mouth. They incubated a small quantity of liver extract, less than enough to produce a remission in human pernicious anemia, with normal gastric juice, and administered the resulting product orally to patients with pernicious anemia. A prompt increase in the circulating red cells and relief of symptoms resulted. They suggest that a different mechanism is involved from that which results after feeding these patients lean beef which has been incubated with normal gastric juice. Fouts and Zerfas[1] likewise report rapid blood cell response and clinical improvement following injections of a similar preparation of a small quantity of liver extract which has been incubated with hog gastric tissue.

Since patients with pernicious anemia apparently must take the specific food throughout the whole of their lives, and liver and stomach eating may grow progressively more distasteful, any further concentration or purification of the active principle is to be watched with interest. The discovery of the relationship of the stomach to blood formation has opened up a new field for physiologic and clinical exploration.

1. Minot, J. R.; and Murphy, W. P.: Treatment of Pernicious Anemia by a Special Diet. J.A.M.A., 87:470, 1926. Editorial, Diet in Pernicious Anemia. Sou. Med. Jour., 20:168, 1927. Liver Extract in Pernicious Anemia. Sou. Med. Jour., 22:414, 1929.

2. Castle, W. B.; Townsend, W. C.; and Heath, C. W.: Observations on the Etiological Relation of Axphylia Gastrica to Pernicious Anemia. Amer. Jour. Med. Sci., 180:305, 1930.

3. Sturgis, Cyrus C.; and Isaacs, Raphael: Desiccated Stomach in Treatment of Pernicious Anemia. J.A.M.A., 93:747, 1929.

4. Morris, R. S.; Schiff, Leon; Burger, George; and Sherman, J. E.: A Specific Hematopoietic Hormone in Normal Gastric Juice. J.A.M.A., 98:1080, 1932. Morris, Roger S.: Erythremia, a Therapeutic Suggestion. J.A.M.A., 101:200, July 15, 1933.

5. Helmer, O. M.; Fouts, Paul J.; and Zerfas, L. G.: Proc. Soc. Exper. Biol. & Med., 30:775, March, 1933.

GLEANINGS FROM RECENT JOURNALS

Denervations: Surgery to Increase Sugar Tolerance.—Operations involving the severing of the nerve supply of particular tissues have increased in popularity in recent years. From many parts of the human body, for example from the colon[2] and ureters,[3] the nerve supply may be cut off without noticeably impairing function.

Various operations, some of them denervations, have been attempted to increase carbohydrate tolerance in diabetes. Crile[4] has denervated the suprarenals in various conditions and notes an increase of sugar tolerance of a small number of diabetic patients after this operation. De Takats and Cuthbert,[5] of Northwestern University Medical School, compared various surgical methods of increasing the sugar tolerance of dogs. Their operations were: ligation of the tail of the pancreas, suprarenal denervation, denervation of the liver, and celiac ganglionectomy. De Takats and Wilder had previously ligated the tail of the pancreas of two diabetic children, and observed that the insulin requirement decreased after operation. This portion of the pancreas contains a large quantity of islet tissue, which shows hypertrophy and hyperplasia after separation from the externally secreting portion of the pancreas. In dogs after ligation, the Chicago investigators observed a rise in sugar tolerance which reached

1. Fouts, P. J.; and Zerfas, L. G.: Liver-gastric Tissue Preparations in the Treatment of Pernicious Anemia. J.A.M.A., 101:188, July 15, 1933.

2. Mitchell, Edward Clay; and Semmes, R. Eustace: Hirschsprung's Disease: Its Pathological Physiology and Apparent Cure of Two Cases Under Observation for Two and Three Years Following Sympathectomy. Sou. Med. Jour., 26:606, July, 1933.

3. Wharton, Lawrence: Denervation of the Ureter: A Clinical Study. Sou. Med. Jour., this issue, p. 677.

4. Danna, J. A.: quoting G. W. Crile in discussion of 4.

5. De Takats, Geza; and Cuthbert, F. P.: Surgical Attempts at Increasing Sugar Tolerance. Arch. Surg., 26:750, May, 1933.

its peak from the third to the sixth month. The ligated tail at first hypertrophies, but later becomes sclerotic and disappears, while the remainder of the pancreas enlarges. An enlargement of the suprarenals was noted in several of the dogs after ligation of the pancreas. When suprarenal denervation was added to the above operation, the sugar tolerance rose and remained high through the period of observation of a year. Denervation of the liver did not affect sugar tolerance. Celiac ganglionectomy raised it to hitherto unobserved levels, and the effect lasted as long as fourteen months unchanged.

The studies are of physiological interest. Obviously, a denervation of any living tissue destroys what it cannot replace. It is to be undertaken only under hopeless conditions, when life has become practically unbearable.

Denervations: Relief of Angina Pectoris.—Surgical removal or destruction of a group of nerves has been employed in clinical practice for relief of the intractable pain of angina pectoris. White, Garrey and Atkins,[1] of Boston, after studies upon the innervation of the heart, have described operative methods which should be effective in control of suffering from this condition. Angina pectoris, they say results from a lack of oxygen in the heart muscle. The interruption of blood flow in the coronary artery causes ischemia of the heart muscle, and an immediate and unmistakable pain response in dogs, which they interpret as angina pectoris. Various nerves were severed by them to demonstrate which pathways were responsible for transmission of this type of cardiac pain. Pain is not interrupted, they say, by severance of the vagus nerves or by sectioning the costal nerves on both sides. Bilateral removal of the sympathetic ganglions from the stellate down through the fourth thoracic ganglion, or section of the upper and posterior dorsal nerve roots, appears to interrupt all sensation from the heart.

For routine treatment of angina pectoris they recommend paravertebral injections of alcohol into the upper four to five thoracic ganglions and their rami. Patients who fail to gain relief by this relatively safe procedure can still be relieved by section of the upper thoracic spinal nerve roots.

Prevention of Embolism After Arsenicals.—Shivers,[2] of the University of Colorado, reports on accidents which have followed the use of arsenicals. There are 800 deaths annually in the United States from these drugs. A large number are due to pulmonary embolism, which occurs, he says, when the reaction of the drug is acid, in which state it is precipitated in the blood. If the *p*H is below 7, neoarsphenamine, arsphenamine, sulpharsphenamine and salyrgan will precipitate in dog serum *in vitro*. Several of the arsenicals precipitate in rabbit's serum *in vivo* if the *p*H is below 7, and produce pulmonary embolism. He outlines a method of adding a very small quantity of an indicator, brom thymol blue, to the solutions to indicate their acidity. If the reaction is as it should be, this dye will be green. If the material has decomposed and has an acid reaction, it will be yellow. He suggests that, since the dye is harmless, it be added to arsenicals as they are put up for clinical use.

Anesthesia in the Ureter.—A depressant action of tribromethylalcohol (avertin) on ureteral musculature is reported by Waddell,[1] of the University of Virginia. The action is the same whether the anesthetic is introduced into the ureter itself or administered intravenously or rectally. The tone and rhythmicity are reduced, and the patency of these muscular tubes is increased. There is no injurious effect and the depressant action disappears as soon as the anesthetic is withdrawn. In urological work, he suggests, this drug may be useful for obtaining relaxation of the ureter or to reduce ureteral spasm.

Secondary Anemia.—The red blood cell is a complex product for the formation of which many food elements are necessary. Anemia may result from lack of vitamin C, or from a prolonged diet of milk which is called "the most nearly perfect food." The liver extract which controls pernicious anemia is not effective in secondary anemia.[1] A liver extract described by Whipple is beneficial in the treatment of anemia from hemorrhage in dogs and, according to Murphy,[2] of Boston, also in human secondary anemia. Murphy has tried various combinations of this liver extract intramuscularly and intravenously, giving it at the same time with several different iron salts by mouth. Liver, he says, agreeing with Whipple, stimulates the formation of the stroma of the red cells and increases their

1. White, J. C.; Garrey, W. E.; and Atkins, J. A.: Cardiac Innervation: Experimental and Clinical Studies. Arch. Int. Med., 26:765, May, 1933.

2. Shivers, G. C.: Pulmonary Embolism from Arsenicals Injected Intravenously: A Method Suggested for Prevention. Arch. Dermat. & Syphilol., 27:901, June, 1933.

1. Waddell, J. A.: Action of Avertin on the Ureter. Jour. Urol., 29:707, June, 1933.

2. Murphy, William P.: Treatment of Secondary Anemia, with Special Reference to the Use of Liver Extract Intramuscularly. Arch. Int. Med., 51:656, May, 1933.

number, without a corresponding pronounced increase in hemoglobin. Iron salts are most valuable in hemoglobin building. After trying a number of iron preparations he prefers ferric ammonium citrate or ferrous carbonate, in an amount to provide 500 mg. of iron daily (ferric ammonium citrate 3 grams daily, ferrous carbonate 5.4 grams). No advantage was offered by intravenous injection.

TWENTY-FIVE YEARS AGO

FROM MEDICAL JOURNALS OF 1908

Clean Advertising.—Dr. Arthur T. McCormack said: "When you understand that five-sixths of the medicinal preparations advertised in the American medical press are fraudulent or worthless, you understand something of the extent of the terrible crime against the sick people of the United States. . . . I would like to impress on the medical profession of Kentucky that there is not the slightest necessity on the face of the earth of paying for a single medical journal you are receiving. You need never pay another subscription and you will get them to the end of time, because they are supported by fraudulent patent medicine advertisements, and these advertisers not only control the advertising pages, but the editorial columns and reading matter from page to page. There are only three medical journals in the United States today outside of the organization journals . . . that have been inaugurated with clean pages. . . . The SOUTHERN MEDICAL JOURNAL of Nashville . . . I hope . . . will receive your support."[1]

Choice of Anesthetics.—Dr. W. M. McCabe said: "Ether is to be preferred to chloroform in cardiac disease, prolonged anesthesia . . . and in all conditions where the patient's vitality is exhausted.

"Chloroform is only preferable to ether in severe bronchial diseases, high arterial tension and atheroma. . . .

"Nitrous oxide may be given with air or oxygen for operations lasting an hour or more."[2]

Pituitary Body.—Dr. J. P. Keller said: "Recently experiments have been made of injecting extracts of the hypophysis cerebri into the circulation, which was followed by a distinct action upon the heart and blood vessels. According to Vincent and Schaffer, we have to do here with two different substances distinguished chemically by their solubility in ether and alcohol, one being soluble and the other not. The first produces a marked fall in blood pressure, while the other increases the pressure and slows and strengthens the heart and produces marked diuresis. . . .

"Curiously enough, Howell and others think these substances do not come from the gland proper, but from the posterior or infundibular portion. Acromegaly has been associated with disease of this body, but I am able to find no record of improvement by means of injecting extracts from the gland in these cases."[1]

In the Interest of Clean Street Car Tickets.—Dr. Alfred Hess,[2] of New York, said: "Street car conductors may be disseminators of tuberculosis and require medical supervision. There should be a rule forbidding a conductor to contaminate the tickets with his saliva. The employment of a chain is advisable, by which to hang his pad."

1. McCormack, Arthur T.: Clean Advertising, address before Kentucky State Medical Association. Sou. Med. Jour., 1: November, 1908.

2. McCabe, W. M.: Anesthesia: Deductions from One Thousand Cases. Sou. Med. Jour., 1:244, 1908.

1. Keller, J. P.: Internal Secretions. Sou. Med. Jour., 1:322, 1908.

2. Hess, A. F.: J.A.M.A., 50:46, 1908.

Richmond—Where We Meet

INTRODUCTION*

The chasm which lay across the nation in the tragic days of 1861 has closed beyond any possibility of future recognition; and the medical association which assembles in Richmond in November is Southern in a geographical sense only. Sectionalism as it was known in that time of dire stress has happily faded with the past so that the American of today, from whatever point of the compass he may hail, is no longer actively conscious of lines of cleavage. Physicians from the North and the South join hands and hearts with the same fine spirit of unity that animates the entire body politic and that is now even softening the humor of those splendid old heroes who once stood eye to eye in mortal combat.

But the memory of that epoch, when the country was riven by war and when the divided manhood of America offered security and life in the defense of principles neither side could passively surrender, can, and should, never be wiped out. And whether one's traditions link him with the Gray or the Blue of that martial period he will for all time find in it, and cherish, a heritage rich in greatness of spirit and self-sacrificing devotion.

*By Dr. Joseph F. Geisinger, Chairman of Sub-Committee on Publicity, General Committee on Arrangements from the Richmond Academy of Medicine, Richmond, Va.

No other city in the United States is as expressive as Richmond of the terrific forces and events of that drama. The War Between the States immediately converted Virginia into a battleground and Richmond became and remained the Capital of the Confederacy—the focal point of the South's memorable effort and equally the goal of Federal assault from every quarter. Here sat statesmen and generals whose names were to be written large upon the pages of history. Roundabout, great armies fought doggedly and the fate of the city hung in the balance while the tide surged back and forth. Finally the dreaded end came and evacuated Richmond lay in ashes.

The story of this chapter in the life of the Convention city of 1933 is told in the following article, written especially for the JOURNAL by Douglas S. Freeman, Ph.D., LL.D., Editor of the Richmond *News-Leader*, and probably the foremost authority in the country on Confederate affairs.

RICHMOND'S CONFEDERATE EPOCH*

The meeting-place of this year's convention of the Southern Medical Association has interest of a sort that no other American city possesses in like measure. Quebec, New York, St. Augustine, New Orleans and Austin recall international rivalries for the possession of America; Richmond is the emblem of the one conflict that threatened the unity that had been achieved after those rivalries had been settled.

The story of the city during the four years of the War Between the States has a drama and a color that accord with the era of tragedy. The coming of the conflict in 1861 found Richmond a quiet city of 37,000 people. Some martial traditions there were of the American revolution, for it was there that Patrick Henry had made the great appeal that culminated with the demand, "Give me liberty or give me death." But the city's atmosphere was essentially cultural and commercial. Her most cherished memories were of John Marshall, of Edgar Allan Poe, and of the notables who had met in public assemblies there. The extension of her trade, by steamship, by railroad and by canal, was her great aim.

Within little more than a month after the secession of Virginia on April 17, 1861, she found herself the capital of the new Confederacy, the objective of the first and greatest efforts of President Lincoln to "restore the Union." Her streets filled with soldiers. Her population rose quickly to 100,000. The fields around her were upturned in frowning trenches. Shops became armories, warehouses were turned into prisons. The great men of the South, gathered there, soldiers and politicians. Thither was brought every rumor and every authentic report of victory or disaster. In her public buildings were held the anxious councils of a new nation. One of her staid residential districts became the "court end" of the town, dominated by the neoclassical residence that was the Gray House of the Confederacy, the counterpart of the White House in Washington.

For a year, the War was kept at a distance from her. Then, in May, 1862, the powerful Army of the Potomac marched up the "Peninsula of Virginia"—the district between the James and the York Rivers—and threatened to lay siege to the city. So close did the enemy come that startled Richmond people could hear the growl of the guns and, from the hilltops and higher buildings at night, could see the red flashes of the opposing artillery. The indecisive battle of Seven Pines, or Fair Oaks, May 31, 1862, brought thousands of wounded to the city, but left the menacing lines where they had been before. General Joseph E. Johnston, the most esteemed of all the Confederate commanders, was wounded in the battle. Wild stories were afloat that the city was to be abandoned. While a frenzied people were passing tense days, General Robert E. Lee, who succeeded General Johnston, was planning an offensive. On June 26, he launched it, and in a seven-day wrestle with General McClellan hurled him back down James River. The loss on the Confederate side was so heavy that the hospitals were overwhelmed and the greatest suffering fell to the lot of the wounded; but the effect on the city was to change apprehension into a confidence that was not shaken until the very end of the War, nearly three years afterwards. Richmond believed that with Lee and the Army of Northern Virginia as her defenders, she could never be taken. She read the battle reports from the Potomac or the Rapidan with profound concern for the wounded, but never with concern for herself.

The White House of the Confederacy in Richmond.

For months, events justified her confidence. McClellan steamed away with his troops to reenforce General John Pope, who was collecting an army around Culpeper. The battles of Second Manassas, Sharpsburg, Fredericksburg, Chancellorsville and Gettysburg followed, all with different outcome, but none of them leaving the enemy within fifty miles of Richmond. General Lee, however, had always to reckon on the possibility that the Union commanders would use their sea-power and would attack Richmond from the South or the East while he was defending her from the North. Largely at his instance, the fortifications of the city were strengthened until they made Richmond a vast entrenched camp, so designed that a few troops, with adequate artillery, could hold the city against odds that otherwise would have been overpowering. The clerks in the government offices, the munition workers

*By Douglas S. Freeman, Ph.D., LL.D., Editor of the Richmond News-Leader, Richmond, Va.

and the boys too young for regular service in the field were organized as "local defense troops," subject to call when the tocsin was sounded in the tower of the old guard-house in the Capitol Square.

At the end of February, 1864, that tocsin sounded ominously. Two Federal columns, one under General Kilpatrick and the other under Colonel Ulric Dahlgren, escaped the vigilance of the Confederate mounted troops and made a raid in Richmond. The city was thinly garrisoned at the time. Only a small force of volunteers and convalescents could be mustered quickly in the threatened suburbs to support the local defense units and a very few batteries of mobile artillery. Kilpatrick, however, was repulsed by artillery, and Dahlgren was driven back in an impetuous charge.

In May, 1864, General Grant crossed the Rapidan at the head of a vast and perfectly equipped army. General Lee repulsed all his direct assaults, but as his adversary kept shifting by the left flank, the Army of Northern Virginia was brought closer to Richmond with every move. On June 1, Grant was back at the old battleground of Cold Harbor, and, though his assaults were hurled back with ghastly losses, the siege of Richmond may be said to have begun at that time. It was not a siege in the sense that the defending force was cut off from the outer world. Railroad communication was preserved, and the farmers from a large area continued to bring their produce to market; but the Federals gradually extended their line until Lee with 45,000 men was holding a front of thirty miles, assailed by more than twice his numbers.

Finally, on April 2, 1865, the line was broken in front of Petersburg, and Richmond had to be evacuated. In a night of wild excitement the government officials and the armed forces left the city. The bridges were burned, the tobacco warehouses were set afire to keep their contents from falling into the hands of the Federals, and a great conflagration swept the business district. The next morning a force of Massachusetts cavalry, vanguard of the conquerors, galloped into the city and hauled down the Confederate and Virginia flags from the capitol. Six days later, the Army of Northern Virginia, reduced to less than 8,000 armed infantry, was forced to surrender at Appomattox courthouse. The Confederate era in Richmond history had ended—in ashes and woe.

A hundred memorials of that tempestuous period will greet the visitors to the convention of the Southern Medical Association. It is said that the scenes of 108 military engagements can be counted from the top of Richmond skyscrapers. The sites of the major actions have been privately purchased and presented to the State. A "battlefield boulevard" now links them so completely that all of them can be visited in an afternoon. The old capitol, the goal of four years of fighting, has been enlarged, but it still preserves the atmosphere of the Confederate epoch and contains many reminders of the men who defended the city. The executive mansion used by President Davis is now a great museum of Confederate relics, containing the sword of Lee, his uniform and his camp equipment, with many relics of "Stonewall" Jackson, Joseph E. Johnston, "Jeb" Stuart, John H. Morgan, Sterling Price and other Confederate leaders, together with much of the official correspondence and many of the effects of President Davis. In the near-by Valentine Museum, itself a fine residence of the period immediately preceding the War, is a remarkable death-mask of Jackson. The Confederate Memorial Institute is adorned with some superb murals of Confederate history, the work of a distinguished French veteran of the World War, Charles Hoffbauer. Many fine monuments to Confederates have places of honor on the streets.

To no visitor is Richmond more interesting than to the physician, especially to the military surgeon. For Richmond had some thirty hospitals during the struggle of the 'sixties. The largest of them, Chimborazo, contained 150 buildings with accommodations for 6,000 patients. It is said to have been the most extensive hospital ever maintained in America prior to the establishment of the Walter Reed in Washington. Now the site of so much human misery—from 60,000 to 70,000 men were treated there—is a spacious park.

Richmond does not live by the past or for the past, but it is a city of memories, memories that stir the heart of all Americans, and most of all the hearts of those whose ancestors bore a part in the struggle that left a united nation to fulfill a nation's destiny.

After Richmond, Washington

TWO BIG WASHINGTON ATTRACTIONS*

Washington has hundreds of attractions which engage the attention each year of something like two million sightseeing visitors. In a single article it is impossible to give any adequate description of these; therefore, I shall devote this space to two of them today, the United States Botanic Garden and the National Zoological Gardens. The United States Botanic Garden, which is at Maryland Avenue between Second and Third Streets, S. W., is one hundred and thirteen years old. Its main new attraction is a conservatory that has just been completed at a cost of one million dollars. The dome of the new conservatory rises to ninety-four feet and to this building many venerable, valuable and huge specimens will be transferred. Many of them are tropical plants.

An idea of the size of the new building may be sensed by the fact that it required three hundred tons of structural steel for the framework. There are thirty-one tons of structural aluminum in the dome, doors and bolts. Eighty-seven thousand cubic feet of glass were necessary to complete this "crystal palace".

There are high-pitched entrance halls level with the outside terrace leading to the main part of the building where will flourish the tallest of the tropical trees and plants. In these trees tropical birds sing and nest. There is in the building a cascade not unlike the famous one of Brussels. Tropical fish will find their homes in the waters of this cascade. The walks in the building are hedged with flower life and interesting plants and shrubs.

Botanists and scientists from all over the world and hundreds of thousands of visitors to the National Capital find special interest in the Botanic Garden.

Among the plants that may be seen in this place are the banyan, the crown of thorns, the tall pine, and the sturdy oak. From the grave of Confucius, Charles A. Dana carried an acorn and planted it in the Botanic Garden, and there it grows today. General

*By Curtis Hodges, Executive Director of the Greater National Capital Committee of the Washington (D. C.) Board of Trade.

Grant planted an acacia, and today the tree grows within a few yards of the great memorial erected to Grant. The Beck-Washington elm is the scion of an elm planted by Washington himself. A plane tree was brought from the Vale of Cashmere and it flourishes; and there are cedars of Lebanon.

Both from the standpoint of history and interest, the United States Botanic Garden is one of the worthwhile sights of Washington.

In one hundred and seventy-six acres of the most beautiful natural park in the world, are the National Zoological Gardens. This show is open every day in the year and all day, and is enjoyed free of cost by millions of visitors each year.

Here is the finest bird house in the world, and the most beautiful reptile house ever built, and there are seven smaller structures which house a collection of twenty-five hundred animals and birds. These National Zoological Gardens have an artistic setting in the hills, valleys, and streams of the seventeen hundred acre primeval Rock Creek Park.

In the new million dollar reptile house, which is always a center of interest, the fourteen foot King

Reptile House at the Zoo.

Cobra is the presiding genius over a priceless collection. Here may be seen also the largest East Indian Python in captivity, the black-hooded cobra of India and the Rainbow Boa, whose name was derived from its exquisite coloring. One of the largest exhibits in the building represents a tropical swamp, inhabited by alligators and crocodiles. At the other end of the building is an interesting group presented to the Zoo by Gifford Pinchot—giant turtles from the Gallapagos Islands, said to be the oldest living reptiles.

The collection of monkeys is one of the finest in the world. The thirty-five species of these human looking animals can always be depended upon to put on one of the best acts in the show for their spectators.

The great Ape and Cat House is the home of Mike and Ike, red-headed baby orangoutangs captured in Sumatra. Here also we find Jojo, one of the oldest apes in captivity and one of the most irascible animals in the whole outfit.

Susan, a young lion, is spoiled and smart. There is a regal looking Bengal tiger, one of the most interesting specimens in captivity. Hymie, the hippopotamus, is one of the most gentle of the Zoo animals. For the great array of animals in this world-known tract, there is a baker who prepares bread and a butcher who supplies the meat. Each year these animals consume approximately one hundred thirty thousand pounds of horse meat, forty thousand pounds of fish, thirty-seven thousand pounds of bread, and two hundred white mice, in addition to other items on the "menu". The elephant eats seventy-five pounds of hay a day, and the bear wants not less than a few dead pigeons, chickens, and from eight to twenty pounds of bread a day.

Visitors in Washington can spend a considerable period looking over the interesting things in the United States Botanic Garden and the National Zoological Gardens.

Book Reviews

A Text-Book of Psychiatry. For students and Practitioners. By D. K. Henderson, M.D., (Edin.), F.R. F.P.S. (Glas.), Physician-Superintendent, The Glasgow Royal Mental Hospital; Lecturer in Psychological Medicine, University of Glasgow, and R. D. Gillespie, M.D., (Glas.), M.R.C.P., D.P.M. (Lond.), Physician for Psychological Medicine, Guy's Hospital, London. Third Edition. 595 pages. New York: The Oxford University Press, 1932. Cloth.

This text book of psychiatry should prove most helpful to the general practitioner, who usually sees the mental patient first.

The three classifications of mental illness are given. The different types of mental disorders, spoken of as types of reaction, are studied in a comprehensive and practical manner. A wealth of case studies are given under the different reaction types. This part of the text is of the standard type.

A valuable innovation to this type of text is the chapter on psychiatry of childhood. The approach to this subject is unique. Child hygienists, welfare workers and all physicians should have some similar work in their library.

Manual of Clinical and Laboratory Technic. By Hiram B. Weiss, A.B., M.D., F.A.C.P., Associate Professor of Medicine, College of Medicine, University of Cincinnati, Cincinnati, Ohio; and Raphael Isaacs, A.M., M.D., F.A.C.P., Associate Professor of Medicine, Assistant Director of the Thomas Henry Simpson Memorial Institute for Medical Research, University of Michigan, Ann Arbor, Michigan. Fourth Edition, reset. 117 pages, with Diet Table. Philadelphia and London: W. B. Saunders Company, 1932. Cloth, $1.50.

The authors have investigated a vast amount of literature and present the results in a brief, comprehensive form. A table on the composition of foods is also included.

Urine and Urinalysis. By Louis Gershenfeld, Ph.M., B.Sc., P.D., Professor of Bacteriology and Hygiene and Director of the Bacteriological and Clinical Chemistry Laboratories at the Philadelphia College of Pharmacy and Science. 272 pages, with 36 engravings. Philadelphia: Lea & Febiger, 1933. Cloth, $2.75.

This volume covers fully and evaluates the tests commonly employed in routine urinalysis, as well as special urinary examinations. It also discusses the structure of the kidneys, kidney function and pathological constituents of the urine.

The Pelvis in Obstetrics. A Practical Manual of Pelvimetry and Cephalometry, Including Chapters on Roentgenological Measurement. By Julius Jarcho, M.D., F.A.C.S., Consulting Gynecologist, Hastings Hillside Hospital; Attending Obstetrician and Gynecologist, Sydenham Hospital. 365 pages with 140 illustrations and 51 tables. New York: Paul B. Hoeber, Inc., 1933. Cloth, $6.00.

The normal pelvis is discussed, emphasis being placed on racial and national variations and statistics of many foreign authors are quoted. Average pelvis measurements for white women are clearly described. Illustrations and diagrams are numerous and good and there is an extensive bibliography. Directions for taking internal pelvic measurements with the instruments of various obstetricians are given in detail. Directions for roentgen ray, pelvimetry and cephalometry are included.

The author regrets the tendency toward neglect of careful pelvic measurements and dependence upon the dangerous test of labor. He is conservative in his stand on the indications for cesarean section. This is an exhaustive study and a valuable reference work.

The International Medical Annual (1933). Fifty-First Year. Carey F. Coombs, M.D., F.R.C.P. (the late), and A. Rendle Short, M.D., B.S., B.Sc., F.R.C.S., Editors. 572 pages. Baltimore: William Wood & Co., 1933. Cloth, $6.00.

The volume contains four new physical signs, a comprehensive review of lung abscess, the new changes in the British pharmacopoeia, a preventive for heat cramp in the use of 0.5 per cent salt water as a beverage, a brilliant summarization of iritis and iridocyclitis, papers on the relief of pain in obstetrics and the radium treatment of cancer to mention only a few of the subjects which are of more common interest. The practitioner will find this an excellent book, not to read from cover to cover, but to be enjoyed from time to time in small doses and for reference.

Clinical Aspects of the Electrocardiogram, Including the Cardiac Arrhythmias. By Harold E. B. Pardee, M.D., Assistant Professor of Clinical Medicine, Cornell University Medical College, New York City. 295 pages, with 74 illustrations. Third Edition, Revised. New York: Paul B. Hoeber, Inc., 1933. Cloth, $5.50.

This book has been extensively revised and in parts rewritten.

There is a brief historical sketch of the development of electrocardiography and a consideration of the physiological origin of the individual deflections of the graph.

There is a complete presentation of the normal EKG, which is one of the most logical and lucid treatises available. The author still uses the maximum heights for the P and R waves adopted in his former editions and does not conform to the criteria for the interpretation of electrocardiograms adopted by the Heart Committee of the New York Tuberculosis and Health Association.

In the chapter describing the operation of the electrocardiograph the Hindle type of machine occupies the greater part.

The two appendices which complete the text are devoted to methods of determining the angle alpha, which include the graphic chart of Dieuaide. The second appendix is a description of methods of filing and indexing records.

The fourth lead of Wolferth and Wood is dismissed in one paragraph.

This is one of the best presentations of the subject available. The illustrations are well chosen and the workmanship well executed.

General Therapeutics of the Practical Medicine Series, 1932. Edited by Bernard Fantus, M.S., M.D., Professor of Therapeutics, University of Illinois College of Medicine; Member, Revision Committee, United States Pharmacopeia and of National Formulary Revision Committee, and Louis B. Kartoon, B.S., M.D., Instructor of Medicine, University of Illinois College of Medicine. 448 pages. Chicago: The Year Book Publishers, Inc., 1933.

During the past year a number of technical advances have been made in the realm of therapeutics, among them being the injection method in the treatment of hydrocele, intercarotid injections of chemical antiseptics and serums for the treatment of brain abscess and meningitis, intravesical injection of mineral oil to relieve pain following cystoscopy and oleothorax. The use of the physically acting laxatives is rapidly becoming popularized. Irritants in the healing of wounds are receiving more mention than usual and the "active principle" of the maggots in treating osteomyelitis is being isolated. The urine has been found to contain a great many secretions and these are elaborately discussed. In the treatment of the psychoses pyretotherapy has become a very useful adjuvant. The practitioner should acquaint himself with these and the other advances that are abstracted in this text.

Urology of the Practical Medicine Series, 1932. Edited by John H. Cunningham, M.D., Associate in Genito-Urinary Surgery, Harvard University Postgraduate School of Medicine. 464 pages, illustrated. Chicago: The Year Book Publishers, Inc., 1933.

The separate volume on urology which makes its appearance this year should be warmly welcomed by the urologist. This change will allow a more complete covering of a field in which the literature is of rapidly increasing volume. Intravenous urography continues to be a leading topic. Transurethral prostatic operations are debated by a number of eminent urologists. There seems to be a broadening of the comprehension, with an inquiring interest in the remainder of the body as related to this special field. The recent congress at Vittel on renal lithiasis is fully reported, giving an excellent survey of this subject.

A Companion to Manuals of Practical Anatomy. By E. B. Jamieson, M.D., Senior Demonstrator and Lecturer on Anatomy, University of Endinburgh. Third Edition. 654 pages. New York: Oxford University Press, 1932. Cloth, $5.00.

This is a compend of anatomy that has now reached its third edition since the first publication twenty years ago. It is a complete revision, since practically every page has changed. Some sections have been entirely rewritten, especially that on the nervous system. Fuller explanations and new material have increased the number of pages by about one hundred. The use of the B.N.A. nomenclature and the improvement in descriptions will enable the anatomy student to make fuller use of this handbook.

Pediatrics of the Practical Medicine Series, 1932. Edited by Isaac A. Abt, M.D., Professor of Pediatrics, Northwestern University Medical School. With the collaboration of Arthur F. Abt, M.D., Assistant in Pediatrics, Northwestern University Medical School. 564 pages. Chicago: The Year Book Publishers, Inc., 1932.

Neonatal death has been discussed by a number of authors. One of the more interesting abstracts is that of an article by Jay I. Durand entitled "Progress in the Feeding of Infants." Among the infectious diseases poliomyelitis evokes continued discussion and thought, convalescent serum as a method of treatment has not yet received unqualified approval. Five cases of vicarious menstruation have been reported during the year in infants under a year, the youngest child being only 3 months. Z. V. Bókay gives five cases in which local meningeal tuberculosis was cured by deep roentgen therapy, a procedure which no doubt will be subjected to more extensive trial.

General Medicine, The Practical Medicine Series, 1932. 837 pages. Chicago: The Year Book Publishers, Inc., 1932.

No startling new discoveries have been announced during the past year; rather there is a general spirit of progressive research and a crystallization of ideas. This volume shows a renewed interest in those diseases that are not spectacular, but which account for a great deal of economic and physical disability as arthritis, encephalitis lethargica and the common cold. Atebrin as a synthetic malaria remedy makes its debut in this issue. Braenuning (p. 177) says: "It amounts to malpractice to say that a lung is healthy without roentgen examination."

Bright's disease is the subject of several essays and the rationale of a moderate protein diet in kidney conditions is elaborated. The anginal symptom continues to be an item of marked interest. The practitioner will find this a valuable review of the field of medicine.

Radiologic Maxims. By Harold Swanberg, B.Sc., M.D., F.A.C.P., Editor of the Radiological Review, Quincy, Illinois. With a foreword by Henry Schmitz, A.M., M.D., LL.D., F.A.C.S., Professor of Gynecology and Head of the Department, Loyola University School of Medicine. 126 pages. Quincy, Illinois: Radiological Review Publishing Company, 1932. Price, $1.50.

The maxims vary in length from a line to a page and each one expresses a general radiological principle. The book will be useful in impressing certain accepted facts upon the student.

Text Book of Materia Medica and Therapeutics. By Sister Alma, Graduate in Pharmacy, Western Reserve University, 1921; Pharmacist and Instructor of Nurses, Mercy Hospital, Canton. 329 pages. New York: The Macmillan Company, 1933. Cloth, $2.50.

The writer touches upon the more important points of materia medica and strives to give the student nurse a working knowledge of drugs that are in daily use. Thirty chapters comprise the text, fifteen hours being allotted for their study. Under a chapter heading, "Internal Antiseptics," serums and vaccines are given as examples. This is a misuse of the term "antiseptic" and should not be countenanced in a text book.

Peripheral Nerve Injuries. By Lewis J. Pollock, M.D., Professor of Nervous and Mental Diseases, Northwestern University, and Loyal Davis, M.D., Professor of Surgery, Northwestern University, Chicago. 678 pages, with 312 illustrations. New York: Paul B. Hoeber, Inc., 1933. Cloth, $10.00.

Dr. Pollock and Dr. Davis have brought peripheral nerve injuries up to date. Ninety-six pages are given to the consideration of the examination of the patient. This subject is well covered and a wealth of illustrations with numerous sensory charts add largely to its practical value.

Differential diagnosis, anatomy, histology and pathology are taken up. The different specific nerve injuries are studied in complete detail. The motor symptoms, sensory symptoms, vasomotor changes, level of the lesions, partial lesions, signs of recovery, differential diagnosis, anatomy, physiology and surgery of each nerve are considered.

The book is profusely illustrated with excellent photographs and charts which add to its practical value. No book of this type which has come to the attention of the reviewer has presented so large a number of valuable illustrations. There are 312 illustrations, chiefly made up of numerous pictures of different views of the same deformity. A bibliography covering almost 2,000 references is an invaluable addition to this subject.

The neurological surgeon, the general surgeon, the industrial surgeon and the orthopedic surgeon can ill afford to be without this monograph.

Intracranial Tumors. By Loyal Davis, M.D., Ph.D. Volume 14 of Annals of Roentgenology, a Series of Monographic Atlases, Edited by James T. Case, M.D., Professor of Roentgenology, Northwestern University Medical School, Chicago. 277 pages, illustrated. New York: Paul B. Hoeber, Inc., 1933. Cloth, $10.00.

This unique monograph is made up of 121 roentgenographic studies and 85 clinical illustrations with a valuable series of brief case studies. It illustrates the extent of cooperation necessary between the neurological surgeon and roentgenologist to perfect the surgeon's diagnostic and operative procedures. However, to quote the author almost verbatim, it demonstrates the fact that intracranial diagnoses cannot be made by mechanical means alone without repeated careful and accurate neurological examinations.

Ventriculography and encephalography are studied in detail as to procedure, selection of procedure, interpretation and value of the studies. Numerous illustrations of ventriculograms and encephalograms are included with illustrations of the gross and microscopic specimens of some of the cases reported.

This book is divided into 9 chapters under the following headings: Introduction, Direct Roentgenological Evidence of Intracranial Tumors, The Meningiomas, The Hypophyseal Adenomas, The Acoustic Neurinomas, The Craniopharyngiomas; The Gliomas, Osteomas, and Angiomas; Ventriculography and Encephalography, and Radiation Therapy of Intracranial Tumors. A bibliography of 322 references is included in this valuable and painstaking study.

The Rise of Preventive Medicine. By Sir George Newman, K.C.B., M.D., F.R.C.P., Chief Medical Officer of the Ministry of Health, University of London, Heath Clark Lectures, 1931, Delivered at the London School of Hygiene and Tropical Medicine. 270 pages with 8 illustrations. New York and London: Oxford University Press, 1932. Price, cloth, $3.00.

The author points out the early origins of medicine in folk lore, magic, customs and religions. He traces the effect of Egypt and the Mosaic law, Greek medicine, the middle ages and the Renaissance with the concurrent rise of physiology upon preventive medicine. To the Greeks he attributes four supreme contributions: "The Ionian concept of the physical elements of the cosmic world of nature; secondly, in the use of these elements—air, water, heat, cold—in the practice of hygiene as a therapeutic means; thirdly, in the vital principles expressed in the Hippocratic corpus; and fourthly, in the application of hygiene to the education and culture of its youth."

In the Seventeenth Century certain clinical observations had been made, but it was in the Eighteenth Century that medicine flourished. In this era Hunter said: "Don't think, try; be patient, be accurate." This remark made to Jenner summarizes the attitude of the profession at this time. With the advent of the mircroscope fresh impetus was given to preventive medicine by the addition of pathology and bacteriology to the armamentarium. This new knowledge often changed empirical into scientific processes.

The last two chapters discuss the application of the discoveries which were outlined and the organization of preventive medicine. The book gives a sufficient though not detailed history of the origins of medicine, emphasis being given to those parts which have a bearing on the field of preventive medicine.

Gastric Anacidity: Its Relations to Disease. By Arthur L. Bloomfield, M.D., Professor of Medicine, Stanford University, San Francisco, California, and W. Scott Polland, M.D., Instructor in Medicine, Stanford University, San Francisco, California. 188 pages. New York: The Macmillan Company, 1933. Cloth, $2.50.

Illustrative of the recent greatly increased interest in this subject, since the observations upon stomach secretion in pernicious anemia, is this volume. It contains valuable statistics of the incidence of anacidity in a large number of diseases.

Southern Medical News

ALABAMA

Dr. Seale Harris, Birmingham, recently gave an address and a clinic at the Oklahoma State Medical Association, Oklahoma City, Oklahoma, and an address before the Washington County Medical Society in Fayetteville, Arkansas.

Dr. F. W. Pickell, formerly of Hope, Arkansas, has located in Brewton.

The annual lecture of the Sigma Xi Club at the University of Alabama, Tuscaloosa, was delivered by Dr. Harry Boyer Weiser, Professor of Chemistry, Rice Institute, Houston, Texas.

The Baldwin County Medical Society, at a recent meeting, made arrangements for the holding of clinics to give medical attention to indigents, these clinics to be held with the assistance of Miss Ada Causey and Miss Mary Quarrels. Dr. F. B. Moore, Fairhope, offered the use of his hospital to further the plan.

Deaths

Dr. Thomas W. Cross, Birmingham, aged 85, died May 3 as the result of injuries received in a fall.

Dr. Michael Joseph Banks, Jacksons Gap, aged 75, died April 4.

ARKANSAS

Dr. Benjamin D. Luck, Pine Bluff, has succeeded Dr. Mahlon D. Ogden, Little Rock, as a member of the State Board of Nurse Examiners.

Dr. Allen B. Jemison, Stuttgart, has succeeded Dr. George A. Hays, resigned, as Director of the Jefferson County Health Department.

Dr. Carl A. Rosenbaum, formerly of Little Rock, has moved to McGehee.

Dr. A. C. Kolb, Hope, has been made Secretary-Treasurer of the Hempstead County Medical Society to fill the unexpired term of Dr. F. W. Pickell.

Dr. F. W. Pickell, formerly of Hope, has moved to Brewton, Alabama.

Dr. W. B. Grayson, formerly of McGehee, has moved to Little Rock, where he will assume his new duties as State Health Officer.

The Tri-County Clinical Society, composed of Hempstead, Clark and Nevada Counties, at a recent meeting elected the following officers for the coming year: Dr. Don Smith, Hope, President; Dr. A. J. Neighbors, Hope, Hempstead County; Dr. Tom Ross, Arkadelphia, Clark County; Dr. Jake Hesterly, Prescott, Nevada County, Vice-Presidents; and Dr. A. L. Buchanan, Prescott, Secretary.

Deaths

Dr. Percy Alexander Riddler, Fort Smith, aged 54, died April 30 of angina pectoris.

Dr. Joseph Freeman Hughes, Smackover, aged 69, died April 2 of cerebral hemorrhage.

Dr. William E. Morriss, Fort Smith, aged 75, died April 3.

Dr. C. K. Campbell, Dover, aged 66, died May 6 of myocarditis and chronic interstitial nephritis.

Dr. Luther Edgar Moore, Searcy, aged 72, died June 4.

Dr. W. M. Brand, Springdale, aged 51, died May 15.

DISTRICT OF COLUMBIA

At a recent meeting of the Medical Society of the District of Columbia an oil painting of Dr. William Gerry Morgan was unveiled and presented to Dr. Morgan as an expression of appreciation of the Society of Dr. Morgan's worth and of his accomplishments for organized medicine. Dr. Thomas A. Groover was Chairman of the Committee in charge of the presentation. Dr. Morgan, in accepting the painting, tendered it to the Society, to remain in the Society's building.

Dr. Edwin A. Merritt, Washington, has been chosen to deliver the second Davidson Foundation Lecture in October, this Foundation being a memorial to Dr. Edward Young Davidson, established in 1929 by the Medical Society of the District of Columbia.

Major James Stevens, Washington, Director of Laboratories and of the Department of Preventive Medicine in the Army Medical School, has been appointed to succeed Major Paul E. McNabb as a member of the National Board of Medical Examiners.

Dr. Arthur D. Wright, Washington, President of the John F. Slater Fund, delivered the address at the tenth commencement of the Saint Philip Hospital School of Nursing, Medical College of Virginia, Richmond, Va.

New officers elected for the scientific sections of the Medical Society of the District of Columbia are: Section on Internal Medicine: Dr. James W. Esler, Chairman, Dr. B. W. Leonard, Vice-Chairman, and Dr. A. J. B. Connolly, Secretary-Treasurer; Section on Ophthalmology and Otolaryngology, Dr. George B. Trible, Chairman, Dr. Ralph S. Pendexter, Vice-Chairman, Dr. Harry F. Davies, Secretary, and Dr. John H. Trinder, Treasurer.

The District of Columbia, Virginia and Maryland Medical Society met at Alexandria, Virginia, recently and elected the following officers for the coming year: Dr. Frank J. Broschart, Gaithersburg, Maryland, President; Dr. C. C. Tumbleson, Sandy Spring, Maryland, Vice-President; Dr. James A. Gannon, Washington, D. C., Recording Secretary; Dr. A. Magruder MacDonald, Washington, D. C., Corresponding Secretary; and Dr. Robert Scott Lamb, Washington, D. C., Treasurer.

Dr. G. A. Wheeler, U. S. Public Health Service, Washington, has been assigned to duty at Spartanburg, South Carolina.

Dr. Joseph W. Mountin, U. S. Public Health Service, Washington, was recently in Richmond and Lawrenceville, Virginia, in connection with studies of and demonstration in rural sanitation.

Dr. C. E. Waller, U. S. Public Health Service, Washington, was in Nashville, Tennessee, recently for a conference on rural sanitation.

Dr. Henry Amiss Hornthal, Washington, and Miss Ellen Joanna Hall were married in May.

DEATHS

Dr. William Clarence Upham, Washington, aged 78, died April 27 of arteriosclerotic heart disease.

Dr. Winthrop Allen Risk, Washington, aged 64, died May 1 of peritonitis as the result of perforated diverticulum of the intestine.

Dr. Floyd McJunkin Allen, Washington, aged 38, died May 22 of pneumonia.

Dr. George W. Kernodle, Washington, aged 72, died May 2 of pneumonia.

Dr. Bruce McVean Mackall, Washington, aged 51, died May 1 of cerebral hemorrhage.

FLORIDA

During the State Association meeting at Hollywood in May an Emory University School of Medicine alumni group held an organization meeting and elected the following officers for the coming year: Dr. L. M. Anderson, Lake City, President; Dr. H. L. Bryans, Pensacola, Vice-President; and Dr. J. C. Pate, Tampa, Secretary-Treasurer.

Dr. E. J. Melville, St. Petersburg, accompanied by Mrs. Melville, recently sailed for Europe, where he will do some work in the clinics of Vienna.

Dr. Jesse Newman McLane, Pensacola, and Miss Marguerite Brittain Thompson were married in May.

Dr. Maurice E. Heck, Miami, has gone to Bushkill, Pennsylvania, where he will practice during the summer.

Dr. Fred H. Albee, of New York, will establish a general hospital at Venice, with the former Parkview Hotel as the main hospital unit.

Dr. Albert H. Freeman, Ocala, will have associated with him in the practice of ophthalmology and otorhinolaryngology Dr. Ralph E. Russell, with offices in the Holder Block.

Dr. Jack Halton, formerly of Sarasota, has located in Tampa, with offices in the Citizens Bank Building.

Dr. W. J. Johnston, Sarasota, has been named Assistant Medical Director of the Florida Medical Center at Venice, which is being established by Dr. Fred H. Albee, of New York, and which will be ready for opening about November 1.

DEATHS

Dr. Wesley Reid Putney, Lake City, aged 45, died May 2 of cerebral hemorrhage.

Dr. Samuel B. Koser, Sebring, aged 67, died April 9 of chronic myocarditis and arteriosclerosis.

Dr. Joseph P. Esch, Daytona Beach, aged 84, died May 9 of pneumonia.

Dr. Thomas Byron King, Gainesville, aged 46, died suddenly May 7 of heart disease.

GEORGIA

The Fifth District Medical Society, at a recent meeting, elected the following officers for the coming year: Dr. Joseph Yampolsky, President; Dr. George W. Fuller, Vice-President; Dr. H. H. Askew, Secretary-Treasurer; and Dr. Edgar Boling, Assistant Secretary-Treasurer; all of Atlanta.

At the Emory University School of Medicine Alumni Clinic Week, held in June, the following officers were elected for the coming year: Dr. John B. Fitts, Atlanta, President; Dr. E. O. Veale, Arnoldsville, First Vice-President; Dr. S. F. Scales, Carrollton, Second Vice-President; and Dr. Marion C. Pruitt, Atlanta, Secretary-Treasurer.

Dr. Albert F. Saunders, Valdosta, is doing postgraduate work in New York.

Postgraduate instruction, with lectures sponsored by the Extension Division of the University of Georgia Medical Department, Emory University School of Medicine, State Board of Health and the Medical Association of Georgia, were given at Valdosta, Statesboro, LaGrange, Athens, and Rome during June and July.

Dr. H. L. Tippins has moved from Savannah to Baxley, where he will practice medicine and surgery.

Dr. Raymond H. King, LaGrange, and Miss Eleanor Haight were married in April.

DEATHS

Dr. William Aaron Gibson, Jr., Thomson, aged 40, died January 27.

KENTUCKY

Dr. Harry Goldberg, Louisville, has moved into new offices in the Breslin Building, where he will practice bone and joint surgery.

Dr. Charles M. Edelen, Louisville, has moved into new offices in the Heyburn Building, and will limit his practice to general surgery.

The Allen County Medical Society has elected the following officers for the coming year: Dr. C. A. Calvert, Scottsville, President; Dr. L. Graves, Scottsville, Vice-President; and Dr. A. O. Miller, Petroleum, Secretary-Treasurer.

The Breathitt County Medical Society has elected the following officers for the current year: Dr. Wilgus Bach, Jackson, President; Dr. Boyd Caudill, Vice-President; and Dr. O. M. Goodloe, Secretary and Treasurer.

The Calloway County Medical Society elected the following officers for the current year: Dr. J. A. Outtond, President; Dr. E. D. Houston, Murray, Vice-President; and Dr. W. H. Graves, Murray, Secretary-Treasurer.

The Letcher County Medical Society has elected the following officers for the current year: Dr. B. F. Reynolds, Carlisle, President; and Dr. T. P. Scott, Carlisle, Secretary.

DEATHS

Dr. John Allen Nelson, Owensboro, aged 70, died April 20 of uremia.

Dr. William Williams, Herndon, aged 87, died March 31 of paralysis of the throat.

Dr. John N. Sebastian, Louisville, aged 58, died March 5.

Dr. Alfred Bradley, Barthell, aged 57, died April 14.

Dr. Fouche Warren Samuel, Louisville, aged 66, died suddenly May 13 of heart disease.

LOUISIANA

The Gorgas Medical Society of New Orleans, organized last year by Dr. Clyde Brooks, met in April at Louisiana University Medical Center for the purpose of awarding honorary fellowships to those "who have distinguished themselves in medical science and research," awards being made to Dr. Arthur Vidrine, Dean of the Medical Center, for his work in surgery; Dr. Joseph Rigney D'Aunoy for his work on electrocardiography; and Dr. Richard Ashman for achievement in research.

Dr. John H. Musser, New Orleans, has been elected Vice-President of the American Medical Association.

Dr. William B. Clark and Dr. Victor C. Smith, New Orleans, have recently moved into new offices in the American Bank Building.

Dr. William B. Clark, New Orleans, was recently appointed local oculist for the Illinois Central Railroad System.

DEATHS

Dr. Henry Dickson Bruns, New Orleans, aged 73, died May 19 of angina pectoris.

Dr. Waldemar Theodore Browne, New Orleans, aged 58, died May 8 of heart disease.

Dr. Louis A. Gaudin, Convent, aged 63, died June 18.

Dr. Louis Murdock, St. Joseph, aged 74, died June 5.

Dr. Emile Regard, Mansura, aged 61, died in an Alexandria hospital on June 3.

MARYLAND

A series of lectures on the History of Medicine has been instituted at the University of Maryland. The first of the series was given by Dr. Henry E. Sigerist, Baltimore, Director of the Institute of the History of Medicine, Johns Hopkins University School of Medicine. Other lectures in the series have been given by Dr. Owsei Temkin and Dr. John Rathbone Oliver, Baltimore, Associates in the History of Medicine, Johns Hopkins University.

Dr. Arthur B. Barrett, Baltimore, was recently installed as President for the sixteenth consecutive term of the Maryland Academy of Medicine and Surgery.

Dr. Adolph Meyer, Baltimore, Director of the Phipps Psychiatric Clinic, Johns Hopkins Hospital, recently spoke before the Royal Medico-Psychological Association in London, delivering the fourteenth Maudsley lecture.

Dr. William H. Wilmer, Baltimore, Director of the Wilmer Institute of Ophthalmology at Johns Hopkins University School of Medicine, recently received from Georgetown University, Washington, D. C., the decoration of the Angelo Secchi Academy of Science. Dr. Wilmer was a member of the faculty of Georgetown University for a number of years.

The Baltimore City Medical Society's Section on Gynecology and Obstetrics recently had as their guest speaker Dr. William

Continued on page 18

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Continued from page 752

Fletcher Shaw, Professor of Gynecology and Obstetrics at the University of Manchester, England.

The Maryland, Virginia and District of Columbia Medical Society, at a recent meeting in Alexandria, Virginia, elected the following officers for the coming year: Dr. Frank J. Broschart, Gaithersburg, Maryland, President; Dr. C. C. Tumbleson, Sandy Spring, Maryland, Vice-President; Dr. James A. Gannon, Washington, D. C., Recording Secretary; Dr. A. Magruder MacDonald, Washington, D. C., Corresponding Secretary; and Dr. Robert Scott Lamb, Washington, D. C., Treasurer.

MISSISSIPPI

Dr. Karl O. Stingily, Meridian, announces the association with him in the Stingily Laboratories of Dr. William Krauss, formerly Professor of Pathology, Bacteriology and Epidemiology of the Medical Department of the University of Tennessee, and late of the Western State Hospital, Bolivar, Tennessee.

Dr. Gettis T. Sheffield is the newly appointed Chief Medical Officer of the Veterans' Administration in Jackson.

Dr. Elmer Gay, Long Beach, is now located at Fort Barrancas, Pensacola, Florida, where he has been assigned to the Medical Staff at the Fort.

Dr. R. D. Dickens has moved from Greenville, where he was associated with Gamble Brothers and Montgomery, to Monticello, Arkansas.

Dr. R. N. Crockett, Winterville, has recently completed a special work on diseases of the heart at the Medical School of the University of Tennessee.

Dr. O. C. Brewer has moved from Eudora to his old home in Southwest Mississippi.

Dr. C. M. Hammond, Walls, recently exhibited his respirator in Memphis.

Dr. L. L. McDougal, Booneville, has resumed his practice after completing a four months' postgraduate course at Tulane University, New Orleans.

Dr. L. W. Long and Dr. Robin Harris, Jackson, held clinics during the June meeting of the Central Medical Society.

The King's Daughters' Hospital Staff of Brookhaven met recently and elected the following officers for the coming year: Dr. J. R. Marquett, President; Dr. G. T. Warren, Vice-President; and Dr. R. S. Savage, Secretary-Treasurer; all of Brookhaven.

Miss Estelle Berryhill is the new Superintendent of Nurses at Northeast Mississippi Hospital.

Dr. J. E. Tate, Tippah County, and Miss Kaye Perry were married in June.

Dr. Joe Evans, Gulfport, President of the Harrison-Stone Hancock Counties Medical Society, and Miss Mary Bliss Herves were married recently.

DEATHS

Dr. Steve Harmon Anderson, Kiln, aged 57, died May 5 of actinomycosis of the scalp and skull.

Dr. J. M. Jenkins, Crenshaw, aged 70, died May 21.

Dr. Ewing Fox Howard, Vicksburg, aged 59, died suddenly June 9.

MISSOURI

A Health Council for Kansas City and Jackson County has been organized with the cooperation of the Council of the Jackson County Medical Society, the Committee on Public Health and Welfare of the Chamber of Commerce, and the Director of Health of Kansas City, the purpose of this Health Council being to coordinate the work of public health agencies for the achievement of efficiency and economy in public health work.

The Missouri State Medical Association, at its annual meeting in Kansas City in May, elected the following officers for the coming year: Dr. C. T. Ryland, Lexington, President-Elect; Dr. J. F. Owens, St. Joseph, Dr. H. J. Wise, Sparta, Dr. P. W. Jennings, Canton, Vice-Presidents; Dr. E. J. Goodwin, St. Louis, reelected Secretary; Dr. E. H. Bartelsmeyer, St. Louis, Assistant Secretary; and Dr. R. A. Woolsey, St. Louis, Treasurer. Dr. W. L. Allee, Eldon, was installed as President for the current year.

Dr. William H. Luedde, St. Louis, Director of the Department of Ophthalmology of the St. Louis University School of Medicine, recently received the Leslie Dana Gold Medal for the most outstanding achievements in the prevention of blindness and the conservation of vision.

The Missouri Society for Mental Hygiene, at its recent annual meeting in St. Louis, elected the following officers for the coming

Continued on page 22

Don't let Fracture Reduction be a Blindfold Test

REDUCING fractures without having the part x-rayed, or without sufficient radiographs, is a hazardous blindfold test. It is impossible to have a complete picture of conditions that exist unless there is adequate radiographic examination in every case.

Radiographs definitely differentiate fractures from sprains. They prevent neglect of a serious condition because superficially it appears to be of minor consequence. Radiographs taken at different angles locate the fracture exactly—show the position of the fragments. Displacements can be corrected with the fewest moves and the least danger of trauma. After reduction, follow-up radiographs prove whether the alignment of the parts is anatomically correct.

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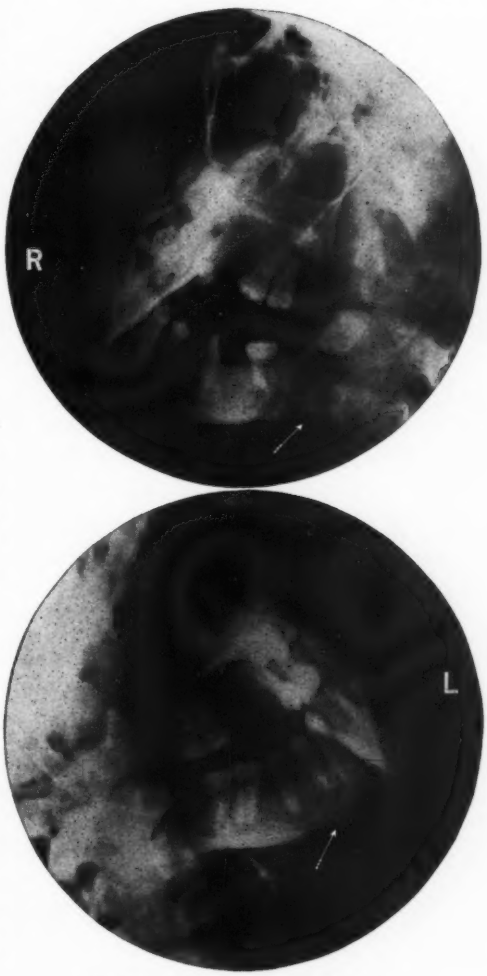

Radiographs of a fractured jaw case

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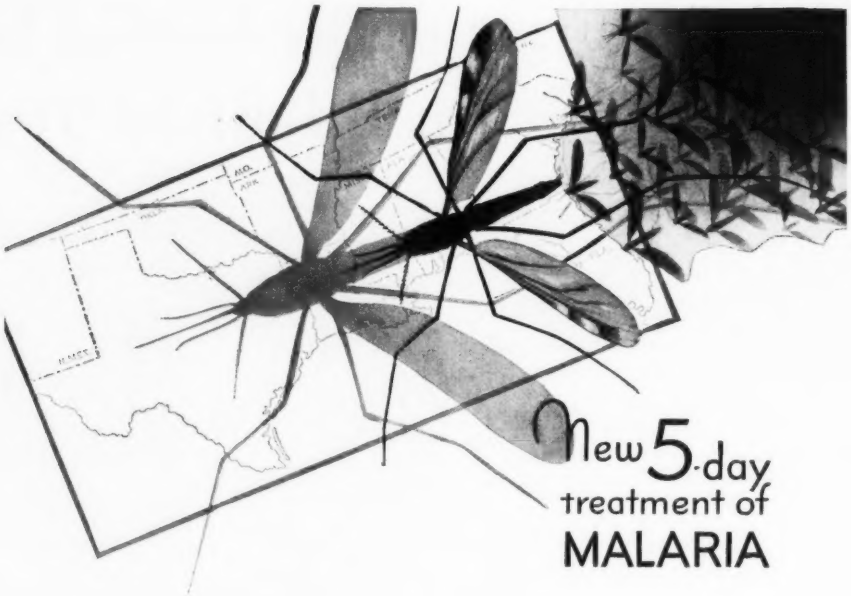

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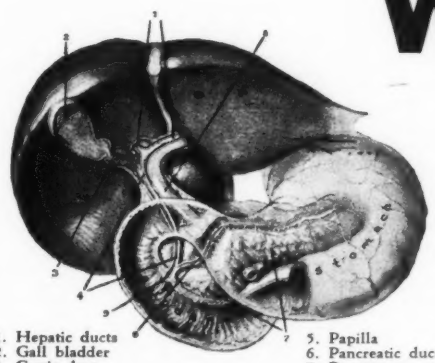

1. Hepatic ducts
2. Gall bladder
3. Cystic duct
4. Common duct
5. Papilla
6. Pancreatic duct
7. Pancreas
8. Portal vein

Pancreas, Common Duct, Portal Vein and Duodenum are shown through the Phantom (transparent) Stomach

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TUCSON, ARIZONA

Continued from page 18

year: Dr. R. C. Fagley, Fulton, President; Dr. Paul Zentay, St. Louis, Vice-President; and Dr. M. A. Bliss, St. Louis, Dr. James Lewald, St. Louis, Dr. William Nelson, St. Louis, Dr. Emmett Hoctor, Farmington, members of the Executive Committee.

Dr. Max A. Goldstein, St. Louis, Director of the Central Institute for the Deaf, received the distinguished service medal of the American Laryngological, Rhinological and Otological Society at its recent annual meeting in Chicago, the award being made for outstanding work in the study and rehabilitation of the deaf child.

Dr. Lewellyn Sale and Dr. W. McKim Marriott, St. Louis, and Rev. Alphonse M. Schwitalla, St. Louis, have been elected Vice-Presidents of the Health and Hospital Department of the Community Council of St. Louis.

Dr. Bransford Lewis, St. Louis, President of the Chess Forum of St. Louis, was invited by the Committee in charge of the chess tournament at the Century of Progress in Chicago to take part in the series of chess games being held there.

Deaths

Dr. Marion Eaton Spurgeon, Red Bird, aged 54, died May 7 in a St. Louis hospital following an operation on the gallbladder.

Dr. James Alexander Campbell, St. Louis, aged 86, died suddenly April 18 of chronic myocarditis.

Dr. Arnold A. Perry, Overland, aged 61, died March 4 of septicemia, following the extraction of a tooth.

Dr. William R. Pennington, St. Joseph, aged 76, died January 17.

Dr. Fred James Hatch, Kansas City, aged 54, died March 31 of retroperitoneal sarcoma.

Dr. David H. Young, Fulton, aged 75, died March 22 of pernicious anemia.

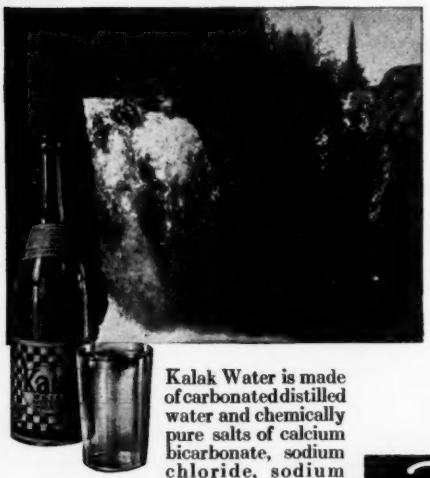
Dr. Clyde Wallace Parsons, Sweet Springs, aged 42, died May 7 of pneumonia.

Dr. Fernando Hardinger Wade, Columbia, aged 68, died April 27 of cerebral hemorrhage and arteriosclerosis.

Dr. Charles Mitchell, St. Louis, aged 64, died May 2 of heart disease.

Continued on page 24

A Pure, Palatable, Carbonated



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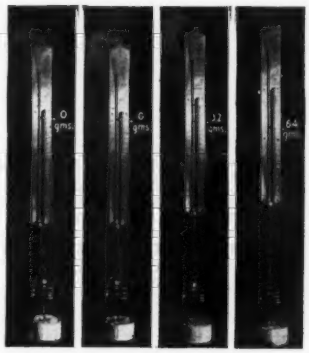

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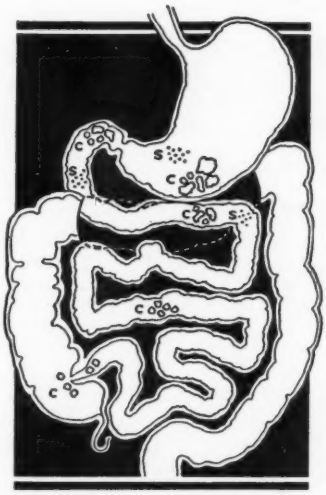

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[1] Morse and Talbot: Diseases of Nutrition and Infant Feeding, pg. 59.
[2] Marriott: Infant Nutrition, pg. 43.

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Continued from page 22

Dr. Frederick Charles Esselbruegge, St. Louis, aged 49, died May 22 of pneumonia following an operation for appendicitis.

Dr. George W. Haverstick, University City, aged 65, died April 30.

NORTH CAROLINA

At the annual meeting of the North Carolina State Medical Society Dr. Paul P. McCain, Sanatorium, was elected President-Elect for the coming year, and Dr. L. B. McBrayer, Southern Pines, reelected Secretary. Dr. Isaac H. Manning, Chapel Hill, is the incoming President.

Dr. William T. Rainey, Fayetteville, and Dr. Sylvester D. Craig, Winston-Salem, were elected members of the State Board of Health at a joint meeting of the State Society and the State Board of Health.

The Tri-State Medical Association of the Carolinas and Virginia, at a recent meeting in Greenville, South Carolina, elected the following officers for the coming year: Dr. Francis B. Johnson, Charleston, South Carolina, President; Dr. Foy Roberson, Durham, North Carolina; Dr. C. N. Wyatt, Greenville, South Carolina; Dr. J. W. Davis, Jr., Lynchburg, Virginia, Vice-Presidents; and Dr. James M. Northington, Charlotte, North Carolina, reelected Secretary-Treasurer.

The thirteenth session of the Southern Pediatric Seminar opened at Saluda, North Carolina, in July, under the leadership of Dr. W. A. Mulherin, Augusta, Georgia.

Dr. L. F. Hall, State Park, is taking postgraduate work in tuberculosis at Saranac Lake, New York.

Dr. John H. Hamilton, Wilmington, has succeeded Dr. Clarence A. Shore, deceased, as Director of the State Laboratory of Hygiene.

Dr. William deB. MacNider, Chapel Hill, Kenan Professor of Pharmacology at the University of North Carolina Medical School, received the honorary degree of Doctor of Science from the Medical College of Virginia during their Commencement exercises in May.

Dr. John Peter Munroe, Charlotte, received an honorary degree from the Duke University at their Commencement exercises.

Dr. Thurman D. Kitchin, Wake Forest, received an honorary degree from the University of North Carolina.

Dr. Charles Preston Mangum, formerly of Kinston, has moved to Richmond, Virginia, for the practice of pediatrics, and will be located at 816 West Franklin Street.

The Sixth District Medical Society, at its annual meeting in Wake Forest recently, elected the following officers for the coming year: Dr. Hubert Haywood, Raleigh, President; Dr. J. S. Bradsher, Stoval, Vice-President; and Dr. Hunter Sweaney, Durham, reelected Secretary-Treasurer.

Dr. J. P. Hunter, Cary, was recently elected Mayor.

Dr. J. M. McAnally has moved from High Point to Reidsville, where he will practice surgery at the Penn Memorial Hospital.

Dr. Lawrence Lincoln Beall, Durham, and Miss Virginia Graham McLean were married in April.

Dr. William C. Mebane, Jr., Wilmington, and Miss Pauline Mae Eckert were married in April.

Dr. John Malloy Clayton Covington, Roanoke Rapids, and Miss Madge Westmoreland Balcome were married in May.

DEATHS

Dr. Lawrence Edward McDaniel, Jackson, aged 52, died May 3 of acute cholecystitis and bronchopneumonia.

Dr. Hurbie Andrews Taylor, Stonewall, aged 42, died April 13 of heart disease.

Dr. Minor Revere Adams, Statesville, aged 78, died suddenly May 27 of heart disease.

Dr. Luther Lafayette Sapp, Reidsville, died April 5.

Dr. C. E. Adams, Gastonia, aged 79, died June 9.

Dr. T. M. Jordan, Raleigh, aged 77, died June 29 in Fayetteville.

Dr. H. E. Austin, Smithfield, aged 45, died May 10 in Coatesville, Pennsylvania.

OKLAHOMA

Dr. LeRoy Long, Oklahoma City, is the newly elected President-Elect of the Oklahoma State Medical Society; and Dr.

Continued on page 26

Phyllicin

the new theophylline salt

Phyllicin is a potent diuretic and cardiac stimulant in doses of 1 tablet two to four times a day, after meals. Prescribe Phyllicin to relieve cardiac distress and reduce edema of cardiac or nephritic origin ...

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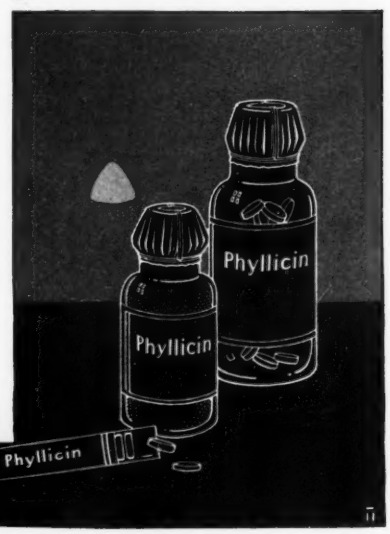

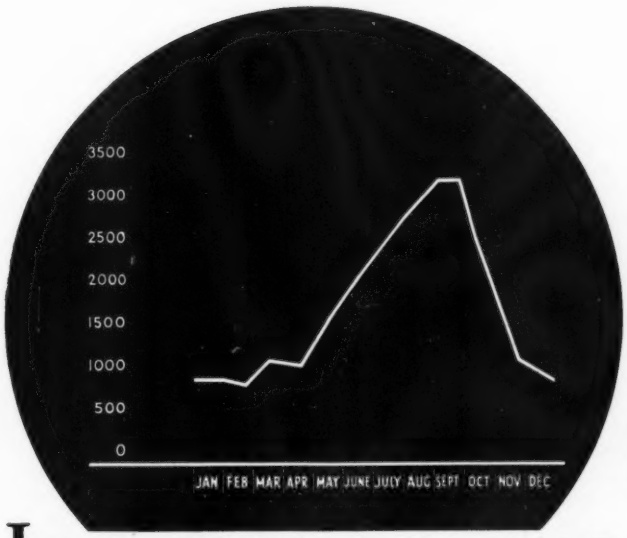

Mortality by months from diarrhea in infancy (under 2 years of age) for 1929 in United States Birth Registration Area. Figures from Bureau of Census Mortality Statistics.

IT IS STILL A FACTOR

Although infant mortality rates have decreased greatly in the past decade, there is still an uptrend during the summer months in the number of deaths caused by intestinal disturbances.

To combat the depletion and to prevent the destruction of body tissue associated with summer diarrhea, infants have immediate need for water, salts and carbohydrates.

Past experience by many physicians has shown the value of the following formula:—

Mellin's Food	**4 level tablespoons**
Water (boiled and cooled)	**16 fluidounces**
Sodium Chloride	**¼ teaspoon**

One to three ounces may be given every hour or two until the stools lessen in number and improve in character. Skimmed milk may be gradually substituted for water until the amount of milk equals the normal quantity. Frequently it may be wise to defer replacing the fat of the milk until after full recovery. In cases where vomiting is a symptom, withholding for a few hours of food and fluids by mouth should precede the introduction of the above formula.

This suggested formula furnishes nutrition well suited to protect the proteins of the body, to prevent rapid loss in weight, to resist the activity of the infectious bacteria, and to assist in the retention of fluids and salts in the body tissues.

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LOUISVILLE, KENTUCKY

Continued from page 24

Tracey H. McCarley, McAlester, was installed as President for the coming year.

Dr. Roy Fisher, Frederick, was graduated in the class of flight surgeons by the School of Aviation Medicine, U. S. Army, at Randolph Field, Texas.

DEATHS

Dr. John Victor Anderson, Fairview, aged 57, died May 7 of encephalitis.

Dr. Frank Edwards Coudert, Guthrie, aged 62, died April 30 of arteriosclerosis and cerebral hemorrhage.

Dr. James A. Dodson, Waukomis, aged 87, died February 23.

Dr. Elmer Forrest Haydon, Tulsa, aged 56, died May 21 of heart disease.

SOUTH CAROLINA

Dr. J. W. Jervey, Greenville, was elected President of the American Laryngological, Rhinological and Otological Society at the recent annual meeting in Chicago.

Dr. C. F. Williams, Columbia, head of the South Carolina State Hospital, was recently elected President of the American Psychiatric Association.

Dr. LeGrande Guerry, Columbia, is the newly elected Vice-President of the American Surgical Association.

Dr. E. V. Valz, recently stationed at Marine Barracks, Paris Island, has been transferred to the U. S. S. Nevada at San Pedro, California.

DEATHS

Dr. James Calhoun Harris, Anderson, aged 74, died May 8 of angina pectoris.

TENNESSEE

Dr. T. J. Stockard, Lawrenceburg, is doing postgraduate work at Vanderbilt University.

Dr. W. R. Irish, South Pittsburg, has moved into a new office at the corner of Elm Avenue and Third Street.

Dr. J. R. Bone, Lebanon, is doing postgraduate work at Vanderbilt University.

Dr. Cecil E. Newell, Chattanooga, and Miss Gretchen Thelen were married in May.

DEATHS

Dr. Oscar Noel Moody, Tennessee City, aged 71, died suddenly April 29 of heart disease.

Dr. Emmett Eugene Pollard, Graysville, aged 39, died April 20 of chronic nephritis.

Dr. Joseph Wilson Bartlett, Nashville, aged 61, died February 25.

Dr. Abram Hassell King, Chestnut Mound, aged 83, died February 16.

Dr. George B. Burrus, Woodland Mills, aged 75, died April 28 in a Jackson hospital.

Dr. Roswell Elmore Flack, Memphis, aged 55, died May 23 of cerebral hemorrhage.

Dr. George Hugh McCain, Whitehaven, aged 66, died May 11 of heart disease.

Dr. A. W. Rudisill, Memphis, aged 65, died May 11.

Dr. Alexander Brown Dancy, Jackson, aged 54, died June 10.

TEXAS

The Texas State Medical Association, at their recent annual meeting, elected the following officers for the coming year: Dr. S. E. Thompson, Kerrville, President-Elect; and Dr. R. H. McLeod, Palestine, Dr. B. C. Smith, Hillsboro, Dr. N. D. Buie, Marlin, Vice-Presidents.

Dr. G. H. De La Perriere has moved from Weslaco to Galveston.

Dr. J. W. Eckhardt has moved from Yoakum to Yorktown.

Dr. A. L. Jennings has moved from Iraan to Abilene.

The Texas Railway Surgeons' Association met in Fort Worth in May and elected the following officers for the coming year: Dr. W. A. Lee, Denison, Persident; Dr. Everett Jones, Wichita Falls, First Vice-President; Dr. M. L. Langford, Mart, Second Vice-President; and Dr. Ross Trigg, Fort Worth, Secretary-Treasurer.

The Texas Dermatological Society met in Fort Worth in May and elected the following officers for the coming year: Dr. Everett C. Fox, Dallas, President; Dr. J. L. Pipkin, San Antonio, Vice-President; and Dr. Everett R. Seale, Houston, reelected Secretary.

Dr. B. F. Payne has moved from Houston to New York City.

Continued on page 28

It's Hot...It's August!

Babies are Suffering from Heat and Summer Complaint

DIARRHEA

Whatever the cause of Diarrhea . . . the water balance must be maintained

It is essential to find a diet that, during the diarrhea, will give sufficient liquid with enough salt to permit a retention of water and provide enough calories to prevent inanition. The purpose of the "transition diet" is to arrest loss of weight, after which a diet is given to increase weight again. (*Révue Francaise de Pediat.*, 1930.)

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The relation between water, fat and other elements of Dryco may be varied to suit the needs of the weakened digestive organs of the sick baby. Its lowered fat content, its flexibility (Dryco may be given in all degrees of concentration and modification) allow of easy regulation of the water to be ingested for a given amount of nutritive elements. *DRYCO is digested and assimilated when other foods fail.*

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Continued from page 26

Dr. L. A. Suggs has moved from San Angelo to Manhattan Beach, California.

Dr. R. W. Taylor has moved from Lujkin to Kerrville.

Dr. H. P. Thomas has moved from Denton to San Antonio.

At an organization meeting in San Antonio of the Theta Kappa Psi Graduate Medical Fraternity the following officers for the coming year were elected: Dr. T. S. Roach, President; Dr. Victor C. Tucker, Vice-President; and Dr. William J. Fetzer, Secretary-Treasurer; all of San Antonio.

The Ector-Midland-Martin-Howard Counties Medical Society has elected the following officers for the coming year: Dr. G. T. Hall, Big Spring, President; Dr. Emmett V. Headlee, Odessa, Vice-President; and Dr. P. W. Malone, Big Spring, Secretary-Treasurer.

The Milam County Medical Society has elected the following officers for the coming year: Dr. Edward Rischar, President; Dr. John Denson, Vice-President; and Dr. G. B. Taylor, re-elected Secretary-Treasurer; all of Camden.

Deaths

Dr. Robert Lee Ramey, El Paso, aged 62, died March 31.

Dr. Horatio G. Lane, Blanket, aged 71, died April 19 of myocarditis and nephritis.

Dr. William H. Hanes, San Angelo, aged 47, died April 30 of pulmonary tuberculosis.

Dr. James Allen Lynch, Pendleton, aged 82, died May 6 of myocarditis.

Dr. S. B. Locker, Menard, aged 57, died March 25.

VIRGINIA

The Virginia, Maryland and District of Columbia Medical Society held its spring meeting at Alexandria in May and elected the following officers for the coming year: Dr. Frank J. Broschart, Gaithersburg, Maryland, President; Dr. C. C. Tumbleson, Sandy Spring, Maryland, Vice-President; Dr. James A. Gannon, Washington, D. C., Recording Secretary; Dr. A. Magruder MacDonald, Washington, D. C., Corresponding Secretary; and Dr. Robert Scott Lamb, Washington, D. C., Treasurer.

Dr. Eugene S. Groseclose, recently of the University of Virginia, has located at Lynchburg and opened offices in the Allied Arts Building for the practice of obstetrics and gynecology.

Dr. Warren T. Vaughan, Richmond, is the newly elected Secretary-Treasurer of the Association for the Study of Allergy.

Dr. Henry C. Davis, formerly of Grundy, has moved to Bluefield.

The Virginia Society of Otolaryngology and Ophthalmology, at a recent meeting in Harrisonburg, elected the following officers for the coming year: Dr. H. Grant Preston, Harrisonburg, President; Dr. Meade Edmunds, Petersburg, Vice-President; and Dr. Edwin W. Burton, Charlottesville, Secretary-Treasurer.

The Alumni Association of the Medical College of Virginia held its annual meeting during commencement week at the College and elected the following officers for the coming year: Dr. John B. Fisher, Midlothian, President; Dr. T. W. Wood (dentist), Richmond, First Vice-President; Dr. H. H. Ware, Richmond, Second Vice-President; and Dr. F. H. Beadles, Richmond, reelected Secretary-Treasurer.

The Northern Neck Medical Association met in Heathsville in May for its semi-annual session.

The James City-New Kent Medical Society met in Toano in June for a dinner and scientific meeting.

Portraits of Dr. Lewis Webb Chamberlayne, first Professor of Materia Medica and Therapeutics, and Dr. Richard L. Bohannan, first Professor of Obstetrics and Diseases of Women and Children, have been presented to the Medical College of Virginia by descendants of these physicians. Portraits were unveiled at exercises during commencement week at the Medical College.

Dr. John P. Snead, Jr., has located at Sperryville after completing a residency at Johnston Memorial Clinic at Abingdon.

Dr. A. W. Armentrout, formerly of Winchester, has moved to Martinsburg, West Virginia.

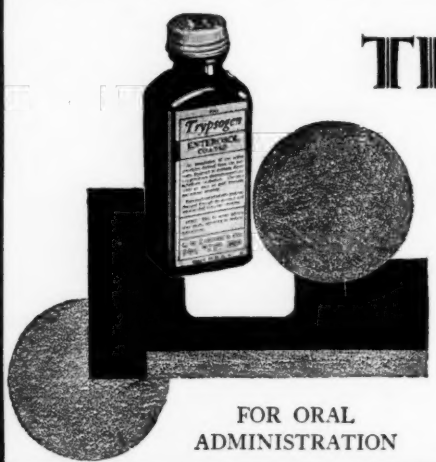
Dr. W. R. Bond, Richmond, Professor of Physiology at the Medical College of Virginia, recently received the decoration of the Purple Heart by the United States War Department in recognition of meritorious service in France during the World War.

Dr. Wyndham Blanton, Richmond, has been awarded the honorary degree of Doctor of Literature by Hampden-Sidney College in recognition of his splendid work in writing the "History of Medicine in Virginia."

Dr. Ira L. Hancock has located at Creeds, after completing a residency at St. Vincent's Hospital, Norfolk.

Continued on page 30

A COMBINATION of all of the active derivatives of the pancreas which control carbohydrate metabolism together with the specific proteins necessary for regeneration of the pancreatic tissues.



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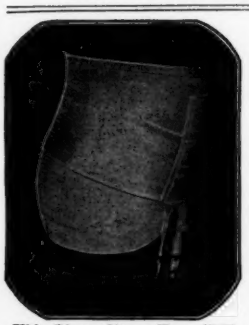

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Continued from page 28

Dr. Micajah Boland has been transferred to the Navy Yard at Portsmouth from the Naval Training Station at Great Lakes, Illinois.

Dr. L. E. Walton, recently of New Market, has moved to Pullman, West Virginia.

Dr. Harry J. Warthen, Jr., Richmond, has returned home to resume his duties after visiting clinics in Germany, Central Europe, England and Scotland.

Dr. Martin D. Delaney, Jr., has located in Alexandria, in the offices occupied by his father, the late Dr. M. D. Delaney.

The Amelia County Medical Society has elected the following officers for the coming year: Dr. H. C. Rucker, Mattoax, President; Dr. J. M. Habel, Jetersville, Vice-President; and Dr. James L. Hamner, Mannboro, Secretary-Treasurer.

The Arlington County Medical Society has elected the following officers for the coming year: Dr. W. C. Welburn, Ballston, President; and Dr. R. N. Sutton, Clarendon, Secretary-Treasurer.

The Norfolk County Medical Society has elected the following officers for the coming year: Dr. W. P. McDowell, President; Dr. C. Carroll Smith, Vice-President; and Dr. Lockburn B. Scott, reelected Secretary-Treasurer; all of Norfolk.

The Alexandria and Fairfax Medical Society held a joint session at Alexandria in May, when they met for a buffet supper and a scientific meeting.

Dr. Robert Preston Hawkins, Jr., Clifton Forge, and Miss Grace Vernon Reynolds were married in June.

Dr. Wilmer Howard Paine, Jr., Charlottesville, and Miss Mildred Goadby Black were married in April.

Dr. Sigmund Newman, Vienna, and Miss Rita Lessner were married in May.

Dr. Wilmore R. Sherrick and Mrs. Helen Crouch Long, both of Richmond, were married recently.

Dr. Hawes Campbell, Jr., and Miss Ellen James Hudgens, both of Richmond, were married in April.

Deaths

Dr. Emmette Edward Walker, Pamplin, aged 73, died June 9.

Dr. John Cleveland Phillips, Portsmouth, aged 48, died recently.

Dr. John Williams Brown, Hampton, aged 59, died June 11.

WEST VIRGINIA

The West Virginia State Medical Association, at its recent annual meeting, elected the following officers for the coming year: Dr. Roy Benn Miller, Parkersburg, President; Dr. Albert G. Rutherford, Welch, Dr. G. H. Barksdale, Charleston, Vice-Presidents; and Dr. T. M. Barber, Charleston, reelected Treasurer.

Dr. H. A. Walkup, Mount Hope, and Dr. O. J. Henderson, Montgomery, were recently honored by being elected to honorary membership in the State Medical Association.

Dr. L. E. Walton has moved to Pullman, coming from New Market, Virginia.

Dr. A. W. Armentrout has located at Martinsburg, and will be connected with the staff of the King's Daughters' Hospital.

Classified Advertisements

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FOR QUICK SALE—Complete Eye, Ear, Nose and Throat equipment and office furnishings. Inventories $5500.00; will sell very cheap for quick cash sale. Inventory furnished on request. Cause of sale, death of owner. Mrs. A. B. Dancy, 28 Northwood, Jackson, Tennessee.

IMMUNITY against diphtheria is produced quickly with Diphtheria Toxoid Mulford (within 6 to 8 weeks instead of 3 to 6 months).

Clinical evidence shows that this protective immunity is developed in 90% to 95% of young children and in 85% to 95% of those over 15 years. In addition, Diphtheria Toxoid Mulford does not contain serum.

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It is supplied in packages of two 1-cc. ampoule-vials (1 immunization); twenty 1-cc. ampoule-vials (10 immunizations); and one 30-cc. ampoule-vial (15 immunizations).

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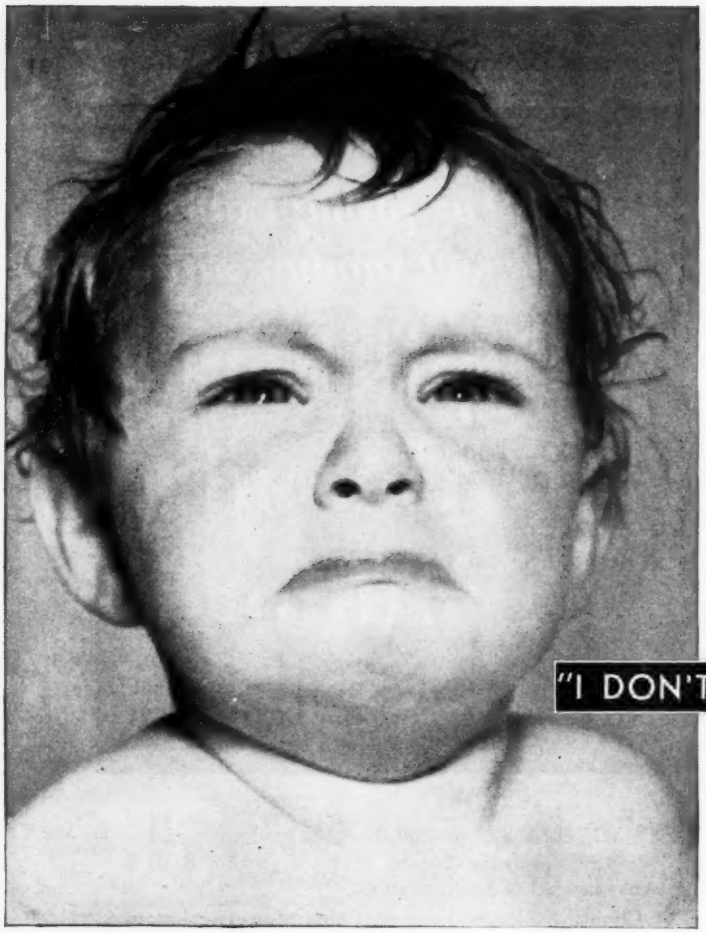

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It takes more severity than many mothers can command to force spinach upon a tearful child. Yet careful menu-planning is needed to make up the 12 mg. of iron required daily. Leichsenring and Flor, as an example, found that children's diets planned to contain 5 and 8.5 mg. iron actually supplied only 3.25 and 6.5 mg., respectively, although the diet was designed to provide a high iron intake and included such foods as raisins, carrots, graham bread, prunes, lettuce, beef, and egg.[1]

PABLUM *tastes good*

AND IS 566% RICHER IN IRON

PABLUM is a food that children really like and take willingly. Added to this virtue, it supplies *known* amounts of iron—more than any other food of equal caloric value! This unique pre-cooked cereal contains 566% more iron than fresh spinach with an iron content of 3.6 mg.[2] (The U. S. Dept. of Agriculture reports an even lower average for spinach—2.5 mg.[3]) When included in the child's daily diet from the third month on, Pablum is a valuable prophylactic against nutritional anemia. Besides the hemoglobin-building element, iron, Pablum contains copper and substantial amounts of calcium, phosphorus, and vitamins A, B, E, and G. Abundant, too, in calories, proteins, fat, and carbohydrates.

1-3 *Bibliography on request.*

For a Delicious Cereal, Just Add Hot Water or Milk (hot or cold)—Pablum Requires No Cooking

Pablum consists of wheatmeal, oatmeal, cornmeal, wheat embryo, yeast, alfalfa leaf and beef bone. Supplies vitamins A, B, E, and G and calcium, phosphorus, iron, copper, and other essential minerals.

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There is Greater Margin of Safety in Dextri-Maltose

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Dextri-Maltose No. 1 (with 2% sodium chloride), for normal babies. Dextri-Maltose No. 2 (plain, salt free), for salt modifications by the physician. Dextri-Maltose No. 3 (with 3% potassium bicarbonate), for constipated babies. "Dextri-Maltose With Vitamin B" for its appetite-and-growth-stimulating properties and the prevention of nutritional anemia (also supplies 2.4 mg. iron and 0.57 mg. copper per oz.).

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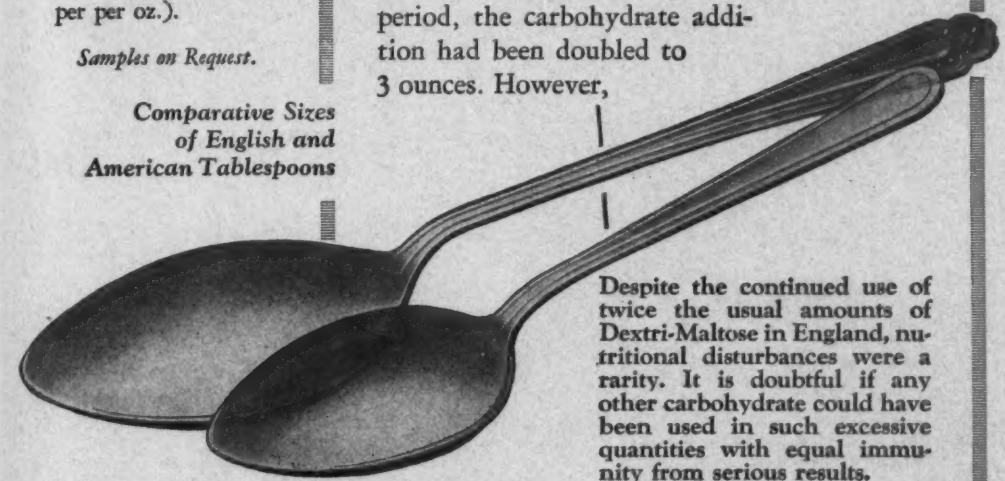
NOTHING tells more graphically the story of greater safety—the freedom from nutritional disturbances in infant feeding that goes with the use of Dextri-Maltose, than the circumstances surrounding its introduction in England.

In England, as in America, it had been prescribed—for modifying cow's milk—by the level tablespoonful.

It was not until Dextri-Maltose had been used in England for over three years, that a prominent English pediatrist pointed out that the British tablespoon is twice the size of the American. The English level tablespoon holds ½ ounce of Dextri-Maltose, the American, ¼ ounce. Where 6 American tablespoonfuls had been prescribed in 24 hours, the infant was actually taking 12, or, in other words, instead of the usual 1½ ounces per 24-hour period, the carbohydrate addition had been doubled to 3 ounces. However,

Comparative Sizes of English and American Tablespoons



Despite the continued use of twice the usual amounts of Dextri-Maltose in England, nutritional disturbances were a rarity. It is doubtful if any other carbohydrate could have been used in such excessive quantities with equal immunity from serious results.

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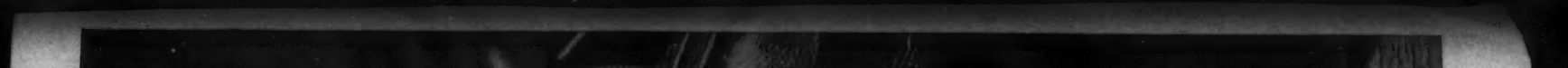
The many emergencies in which it is urgently needed—traumatic shock, apparent death, anaphylaxis, serum reactions, and asthmatic paroxysms—suggest the wisdom of always keeping a supply of Adrenalin* in the emergency bag.

Many clinicians with wide experience in immunization work inject Adrenalin preceding or with the injection of biologicals or other substances containing foreign proteins, in cases where the patient is suspected of being subject to allergic reaction. This simple precautionary measure may prevent allergic reaction and may be the means of preventing a serious or even fatal protein shock.

A supply of Adrenalin ampoules in your office and in your emergency bag not only provides a means of preventing allergic reactions, but may enable you to administer life-saving medication in an emergency.

Adrenalin Chloride Solution 1:1000 is available in one-ounce bottles and in boxes of one dozen and one hundred 1-cc. ampoules (Ampoule No. 88).

***The Parke-Davis brand of Epinephrine, U. S. P.**

PARKE, DAVIS & COMPANY

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